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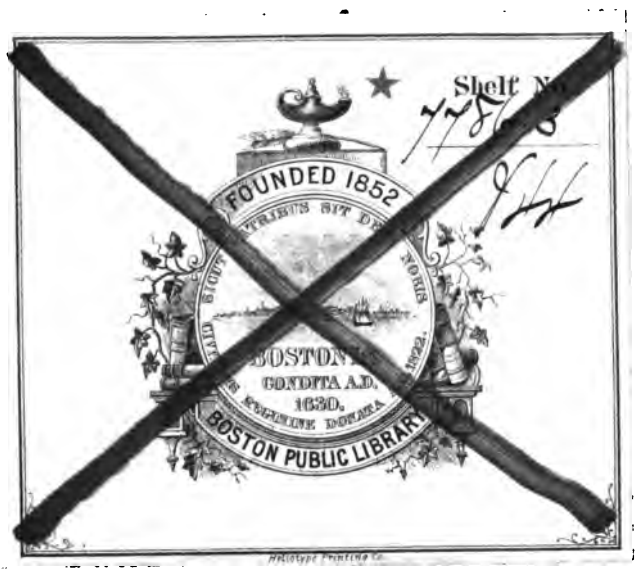

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LONDON:
SMITH, ELDER & CO., 15, WATERLOO PLACE.
1893.

B. H.
March 6. 1894
A.
Ir. Cont.

THE present publication, being the Forty-fourth Volume of Transactions, constitutes the Forty-seventh published Annual Report of the Pathological Society's Proceedings.

The COUNCIL think it right to repeat that the exhibitors are alone responsible for the descriptions given of the Specimens exhibited by them, the only change made in the Reports furnished by the authors being such verbal alterations as were absolutely necessary.

20, HANOVER SQUARE, W.;
October, 1893.

* 7786.3
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Presidents of the Society.

ELECTED

1846 CHARLES J. B. WILLIAMS, M.D., F.R.S.
1848 CHARLES ASTON KEY.
1850 PETER MERE LATHAM, M.D.
1852 CÆSAR H. HAWKINS, F.R.S.
1853 BENJAMIN GUY BABINGTON, M.D., F.R.S.
1855 JAMES MONCRIEFF ARNOTT, F.R.S.
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1861 JAMES COPLAND, M.D., F.R.S.
1863 SIR PRESCOTT G. HEWETT, BART., F.R.S.
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1867 SIR JOHN SIMON, K.C.B., D.C.L., F.R.S.
1869 SIR RICHARD QUAIN, BART., M.D., LL.D., F.R.S.
1871 JOHN HILTON, F.R.S.
1873 SIR WILLIAM JENNER, BART., M.D., K.C.B., D.C.L., F.R.S.
1875 GEORGE D. POLLOCK.
1877 CHARLES MURCHISON, M.D., LL.D., F.R.S.
1879 JONATHAN HUTCHINSON, F.R.S.
1881 SAMUEL WILKS, M.D., F.R.S.
1883 JOHN WHITAKER HULKE, F.R.S.
1885 JOHN SYER BRISTOWE, M.D., F.R.S.
1887 SIR JAMES PAGET, BART.. D.C.L., LL.D., F.R.S.
1889 WILLIAM HOWSHIP DICKINSON, M.D.
1891 SIR GEORGE MURRAY HUMPHRY, M.D., LL.D., F.R.S.
1893 FREDERICK WILLIAM PAVY, M.D., F.R.S.

OFFICERS AND COUNCIL

OF THE

Pathological Society of London,

ELECTED AT

THE GENERAL MEETING, MAY 16TH, 1893,

FOR THE SESSION 1893–94.

President.

FREDERICK WILLIAM PAVY, M.D., F.R.S.

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Treasurer.

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HOWARD HENRY TOOTH, M.D.	C. B. LOCKWOOD.
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DAWSON WILLIAMS, M.D.	SAMUEL G. SHATTOCK.
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GEORGE NEWTON PITT, M.D.
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SEYMOUR J. SHARKEY, M.D.
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W. WATSON CHEYNE.
HENRY HUGH CLUTTON.
FREDERICK S. EVE.
ROBERT WILLIAM PARKER.
SAMUEL G. SHATTOCK.

*** *Members are requested to inform the Secretaries of any corrections which may be necessary.*

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

BILLROTH, THEODOR, M.D., Professor of Surgery in the University of Vienna.
CHAUVEAU, A., M.D., Professor of Physiology at the Medical School of Lyons.
VON HELMHOLTZ, H., M.D., Professor of Physiology in the University of Heidelberg.
JENNER, SIR WILLIAM, Bart., M.D., K.G.C.B., D.C.L., LL.D., F.R.S., Greenwood, Bishop's Waltham, Hants.
LUDWIG, C., M.D., Professor of Physiology in the University of Leipzig.
METCHNIKOFF, E., M.D., Directeur de l'Institut Pasteur, Paris.
PASTEUR, PROFESSOR L., Member of the Institute, Paris.
RINDFLEISCH, EDOUARD, M.D., Professor of Pathological Anatomy in the University of Bonn.
SIMON, SIR JOHN, K.C.B., D.C.L., LL.D., F.R.S., 40, Kensington-square, W.
THIERSCH, CARL, M.D., Professor of Surgery in the University of Leipzig.
VIRCHOW, RUDOLF, M.D., Professor of Pathological Anatomy in the University of Berlin.

EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.	V.-P.—Vice-President.
Pres.—President.	S.—Secretary.
T.—Treasurer.	C.—Member of Council.

Those marked thus (L) have paid Composition Fee for Annual Subscription.
Those marked thus (‡) have paid Composition Fee for Transactions.

GENERAL LIST OF MEMBERS.

Elected

1891 ABBOTT, FRANCIS CHARLES, M.D., St. Thomas's Hospital Medical School, S.E.

L1879 ABERCROMBIE, JOHN, M.D., 23, Upper Wimpole-street, W.

1885 ABRAHAM, PHINEAS S., M.A., M.D., 2, Henrietta Street, W.

1858 ACLAND, Sir HENRY WENTWORTH, Bart., K.C.B., M.D., F.R.S., Broad-street, Oxford.

1883 ACLAND, THEODORE DYKE, M.D., 74, Brook-street, W. (C. 1892-3.)

1891 ADAMI, J. GEORGE, M.A., M.B., Jesus College, Cambridge.

‡1866 ADAMS, ARTHUR BAYLEY.

1890 ADAMS, JAMES, 4, Chiswick-place, Eastbourne.

L.O.M. ADAMS, WILLIAM, 5, Henrietta-street, Cavendish-square, W. (C. 1851-4. V.-P. 1867-9.)

L1848 AIKIN, CHARLES A., 12, Ladbroke-terrace, W. (C. 1864-6.)

1872 AIKIN, CHARLES EDMUND, 12, Ladbroke-terrace, W.

L1882 ALLCHIN, WILLIAM HENRY, M.B., 5, Chandos-street, W.

1877 ALTHAUS, JULIUS, M.D., 48, Harley-street, W.

1884 ANDERSON, ALEXANDER RICHARD, 5, East Circus-street, Nottingham.

1868 ANDERSON, J. FORD, M.D., 41, Belsize-park, N.W.

1871 ANDERSON, WILLIAM, 2, Harley-street, W. (C. 1888-90.)

L1863 ANDREW, JAMES, M.D., Sunnycote, Emery Down, Lyndhurst, Hants. (C. 1868-70. V.-P. 1889-90.)

1887 ARKLE, CHARLES, M.D., 66, Wimpole-street, W.

1883 ASHBY, HENRY, M.D., 13, St. John-street, Manchester.

1882 AXE, J. WORTLEY, College House, Great College-street, N.W.

1863 BAGSHAWE, FREDERICK, M.A., M.D., 16, Warrior-square, Hastings.

‡1856 BALDING, DANIEL BARLEY, Royston, Herts.

1881 BALLANCE, CHARLES A., M.S., 106, Harley-street, W. (C. 1890-2.)

1875 BARKER, ARTHUR E. J., 87, Harley-street, W. (C. 1884-6.)

1885 BARLING, GILBERT, M.B., 85, Edmund-street, Birmingham.

1874 BARLOW, THOMAS, M.D., B.S., 10, Wimpole-street, W. (C. 1879-81.)

1862 BARRATT, JOSEPH GILLMAN, M.D.

L1877 BARROW, A. BOYCE, 37, Wimpole-street, W.

1881 BARRS, ALFRED GEORGE, M.D., 22, Park-place, Leeds.

1879 BARTLETT, HENRY, M.D., Defoe-road, Tooting-corner, S.W.

L1853 BARWELL, RICHARD, 55, Wimpole-street, W. (C. 1862-4. V.-P. 1889-90.)

L1861 BASTIAN, H. CHARLTON, M.A., M.D., F.R.S., 8A, Manchester-square, W. (C. 1869-71. V.-P. 1885-7.)

1877 BATEMAN, ARTHUR W., B.A., Tenterfield, New South Wales.

Elected

L1876 BATTESON, JOHN, 26, Windsor-road, Forest-gate, E.

1882 BATTLE, WILLIAM HENRY, 2, Mansfield-street, W.

1870 BÄUMLER, CHRISTIAN G. H., M.D., University of Erlangen.

1874 BEACH, FLETCHER, M.B., Two Elms, Chislehurst-road, Sidcup, Kent.

1892 BEADLES, CECIL F., Colney Hatch Lunatic Asylum, N.

1879 BEALE, EDWIN CLIFFORD, M.B., 23, Upper Berkeley-street, W.

1852 BEALE, LIONEL S., M.B., F.R.S., 61, Grosvenor-street, W. (C. 1858-9. V.-P. 1874-5.)

1856 BEALEY, ADAM, M.D., M.A., Felsham Lodge, Hollington-park, St. Leonard's-on-Sea.

1865 BEEBY, WALTER, M.D., Bromley, Kent.

L1880 BEEVOR, CHARLES EDWARD, M.D., 33, Harley-street, W. (C. 1888-90.)

1883 BENHAM, ROBERT FITZROY, Abercorn House, Castletown-road, West Kensington, W.

1886 BENNETT, FREDERICK JOSEPH, 24, George-street, Hanover-square, W.

1877 BENNETT, WILLIAM HENRY, 1, Chesterfield-street, W. (C. 1891-93.)

1889 BENTLEY, ARTHUR, J.M., M.D.

1878 BERNARD, FRANCIS R., M.D.

1882 BERRIDGE, WILLIAM ALFRED, Redhill, Surrey.

1886 BERRY, JAMES, 60, Welbeck-street, W.

1891 BEVILLE, FREDERICK WELLS, The Firs, Palace-road, East Molesey.

‡1856 BICKERSTETH, EDWARD R., 2, Rodney-street, Liverpool.

1889 BIDWELL, LEONARD ARTHUR, 54, Harley-street, W.

1882 BINDLEY, PHILIP HENRY, M.B., Branksome-road, St. Leonard's-on-Sea.

1890 BINDLEY, ROBERT ALFRED, Inter. M.B., 35, Highbury-hill, N.

1878 BINDON, WILLIAM JOHN VEREKER, M.D., 18, St. Ann's-street, Manchester.

L1850 BIRKETT, EDMUND LLOYD, M.D., Westbourne Rectory, Emsworth, Hampshire. (C. 1856-7.)

1881 BISS, CECIL YATES, M.D., 135, Harley-street, W.

1865 BISSHOPP, JAMES, Bedford-place, Tunbridge Wells.

1877 BLACK, JAMES, Hillside, Hermitage-road, Norwood.

1889 BLACK, ROBERT, M.D., 6, Pavilion Parade, Brighton.

1850 BLAGDEN, ROBERT, Stroud, Gloucestershire.

1863 BLANCHET, JEAN B., M.D., M.S., Montreal, Quebec, Canada.

1876 BLASSON, WILLIAM, 23, Seymour-street, W.

1879 BOILEAU, J. P. H., M.D., Brigade-Surgeon, Army.

1876 BOND, THOMAS, M.B., 7, Broad Sanctuary, Westminster, S.W.

1869 BOURNE, WALTER, M.D. (Travelling).

1861 BOWER, RICHARD NORRIS.

1881 BOWLBY, ANTHONY A. (HON. SECRETARY), 24, Manchester-square, W. (C. 1886-8. S. 1893.)

1892 BOYCE, RUBERT WILLIAM, M.B., 30, Sussex-square, Brighton.

1882 BOYD, STANLEY, M.B., 134, Harley-street, W. (C. 1893.)

1889 BRADFORD, JOHN ROSE, M.B., 52, Upper Berkeley-street, W.

1883 BRADSHAW, JAMES DIXON, M.B., 30, George-street, Hanover-square, W.

Elected

1880 Bramwell, Byrom, M.D., 23, Drumsheugh-gardens West, Edinburgh.
1889 Bredin, J. Noble, 1, Norton Folgate, E.
1877 Bridges, Robert, M.B., M.A., Manor House, Yattendon, Berks.
‡1867 Bridgewater, Thomas, LL.D. Glas., M.B. Lond., Harrow-on-the-Hill, Middlesex.
1873 Briggs, Jacob Myers, M.D., Coeymans, New York, U.S.
1868 Bright, George Charles, M.D., Cannes, Alpes Maritimes, France.
1857 Briscoe, John, 5, Broad-street, Oxford.
1885 Briscoe, John F., Rawdon House, Alton, Hants.
1892 Bristow, George Hamilton, 182, Stockwell-park-road, S.W.
L1851 Bristowe, John S., M.D., F.R.S., 13, Old Burlington-street, W. (C. 1854-8. S. 1861-4. C. 1865-7. V.-P. 1868-76, 1887. P. 1885-6.)
1860 Broadbent, Sir William Henry, Bart., M.D., 84, Brook-street, W. (C. 1871-3. V.P. 1882-4.)
1886 Brockatt, Andrew Alexander, St. Cuthbert's, Malvern.
L1852 Brodhurst, Bernard E., 20, Grosvenor-street, W. (C. 1862-4.)
1884 Brodie, Charles Gordon, 30, Harley-street, W.
1863 Brodie, George Bernard, M.D., 3, Chesterfield-street, Mayfair, W.
L1865 Brown, Augustus, M.D., 5, Clermont-terrace, Preston-park, Brighton.
1871 Brown, Frederick Gordon, 17, Finsbury-circus, E.C.
1875 Browne, George Buckston, 80, Wimpole-street, Cavendish-square, W.
L1866 Browne, Lennox, 15, Mansfield-street, W.
1877 Bruce, J. Mitchell, M.D., 70, Harley-street, W.
1890 Brunton, T. Lauder, M.D., D.Sc., LL.D., F.R.S., 10, Stratford-place, W.
L1855 Bryant, Thomas, 65, Grosvenor-street, W. (C. 1863-6. V.-P. 1877-9.)
L1854 Buchanan, Sir George, M.D., LL.D., F.R.S., 27, Woburn-square, W.C. (C. 1864-6. V.-P. 1880-2.)
1890 Buckland, Francis O., M.A., M.B., C.M., 6, Lower Sloane-street, S.W.
1891 Burghard, Frederic Francois, M.D., M.S., 46, Weymouth-street, W.
1878 Burnet, Robert William, M.D., 6, Upper Wimpole-street, W.
1880 Burton, Samuel Herbert, M.B., Norfolk and Norwich Hospital, Norwich.
1887 Butler-Smythe, Albert Charles, 76, Brook-street, W.
1872 Butlin, Henry Trentham (C.), 82, Harley-street, W. (C. 1876-8, 1887-9. S. 1884-6. V.-P. 1891-2.)
1866 Butt, William Frederick.
L1883 Buxton, Dudley W., M.D., 82, Mortimer-street, W. (9, Portland-place, W.)
L1856 Buzzard, Thomas, M.D., 74, Grosvenor-street, W. (C. 1869-70. V.-P. 1881-3.)

1893 Caddy, Duncan James, M.B., 24, Earl's Court-gardens, S.W.
1885 Cahill, John, 12, Seville-street, Lowndes-square, S.W.
1892 Campbell, Henry Johnstone, M.D., 6, St. Thomas's-street, S.E.

Elected

1891 CARLESS, ALBERT, M.S., M.B., 10, Welbeck-street, W.

1891 CARR, JOHN WALTER, M.D., 40, Bloomsbury-square, W.C.

1855 CARTER, H. VANDYKE, M.D., Scarborough.

1876 CARTER, ROBERT BRUDENELL, 27, Queen Anne-street, W.

1879 CASSIDY, JOSEPH LAMONT, M.D., 44, Harley-street, W.

1877 CASSON, JOHN HORNSBY, Teheran, Persia.

L1868 CAVAFY, JOHN, M.D., 2, Upper Berkeley-street, W. (C. 1881-3.)

1864 CAY, CHARLES VIDLER, Deputy Surgeon-General, Army.

1863 CAYLEY, WILLIAM, M.D. (TREASURER), 27, Wimpole-street, W. (C. 1870-1, 1875-8. S. 1872-4. V.-P. 1884-6. T. 1888-93.)

1869 CHAFFERS, EDWARD, Keighley, Yorkshire.

1884 CHAFFEY, WAYLAND CHARLES, M.D., 13, Montpellier-road, Brighton.

1891 CHAPLIN, ARNOLD, M.B., 24, Finsbury-circus, E.C.

1876 CHARLES, T. CRANSTOUN, M.D., M.C., 2, Edinburgh Mansions, Victoria-street, S.W.

1884 CHAVASSE, THOMAS FREDERICK, M.D., C.M., 24, Temple-row, Birmingham.

1879 CHEYNE, WILLIAM WATSON, M.B., C.M., 75, Harley-street, W. (C. 1885-7. V.-P. 1892-3.)

1858 CHILD, GILBERT W., Cowley House, Oxford.

1873 CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales [care of Messrs. Dawson, 121, Cannon-street, E.C.].

1865 CHURCH, WILLIAM SELBY, M.D., 130, Harley-street, W. (C. 1871-3.)

L1868 CHURCHILL, FREDERICK, M.B., 4, Cranley-gardens, Queen's-gate, S.W.

L1861 CLAPTON, EDWARD, M.D., 22, St. Thomas's-street, Southwark, S.E.

1854 CLARK, Sir ANDREW, Bart., M.D., LL.D., F.R.S., 16, Cavendish-square, W. (C. 1862-5. V.-P. 1881-3.)

1872 CLARK, ANDREW, 71, Harley-street, W.

1886 CLARK, FRANCIS WILLIAM, The Dispensary, Newcastle-on-Tyne.

1883 CLARKE, ERNEST, M.D., B.S., 41, Lee-terrace, Blackheath, S.E.

1891 CLARKE, J. JACKSON, M.B., St. Mary's Hospital, W.

1885 CLARKE, JOHN MICHELL, M.B., 28, Pembroke-road, Clifton, Bristol.

1881 CLARKE, W. BRUCE, M.B., 46, Harley-street, W. (C. 1892-3.)

L1875 CLUTTON, HENRY HUGH, M.A., 2, Portland-place, W. (C. 1884-6. V.-P. 1892-3.)

‡1865 COATES, CHARLES, M.D., 10, Circus, Bath.

1885 COATS, JOSEPH, M.D., 31, Lynedoch-street, Glasgow.

1856 COCKLE, JOHN, M.D., M.A., 5, Suffolk-place, S.W.

1892 COLE, ROBERT HENRY, Moorcroft, Hillingdon, Uxbridge.

1886 COLLIER, WILLIAM, M.D., 62, High-street, Oxford.

1891 COLLINS, EDWARD TREACHER, 47, Devonshire-street, W.

1888 COLLINS, WILLIAM JOB, M.D., M.S., 1, Albert-terrace, Regent's-park, N.W.

1878 COLLYNS, R. T. POOLE, 20, Lingfield-road, Wimbledon.

1888 COLMAN, WALTER STACY, M.B., 41, Torrington-square, W.C.

Elected

1882 COLQUHOUN, DANIEL, M.D., Dunedin, New Zealand.
1891 COOK, HERBERT G. GRAHAM, Victoria Hospital, Chelsea, S.W.
1858 COOKE, R. T. E. BARRINGTON, 15, St. Nicholas-cliff, Scarborough, Yorkshire.
1871 COOKE, THOMAS, 40, Brunswick-square, W.C.
1866 COOMBS, ROWLAND HILL, Mill-street, Bedford.
1892 COOPER, C. DUDLEY, 171, Stanhope-street, N.W.
1875 CORY, ROBERT, M.D., 73, Lambeth Palace-road, S.E.
1892 COTTERELL, EDWARD, 5, Halkin-street West, S.W.
1876 COTTLE, WYNDHAM, M.D., 3, Savile-row, W.
L1861 COUPER, JOHN, 80, Grosvenor-street, W. (C. 1870-2.)
1873 COUPLAND, SIDNEY, M.D., 16, Queen Anne-street, W. (C. 1878-81, 1889-91. S. 1886-8. V.-P. 1892-3.)
1884 CRICHTON, GEORGE, M.B., 3, Cambridge-villas, Twickenham.
1873 CRIPPS, WILLIAM HARRISON, 2, Stratford-place, W. (C. 1883-5. V.-P. 1893.)
L1877 CROCKER, HENRY RADCLIFFE, M.D., 121, Harley-street, W. (C. 1887-9.)
1856 CROFT, JOHN, 6, Mansfield-street, W. (C. 1870-2. V.-P. 1882-4.)
1879 CROOKE, GEORGE FREDERICK, M.D., 2, Edmund-street, Birmingham.
1886 CROOKSHANK, EDGAR, M.B., Saint Hill, near East Grinstead, Sussex. (C. 1890-3.)
1861 CROSBY, THOMAS BOOR, M.D., 21, Gordon-square, W.C.
1875 CROSS, FRANCIS RICHARDSON, 5, The Mall, Clifton, Bristol.
1890 CROWLE, THOMAS H. RICKARD, 3, Campden Hill-road, W.
1889 CUFF, ROBERT, M.B., Huntriss-row, Scarborough.
1885 CULLINGWORTH, CHARLES JAMES, M.D., 46, Brook-street, W.
1871 CUMBERBATCH, ELKIN, 17, Queen Anne-street, W.
1873 CURNOW, JOHN, M.D., 3, George-street, Hanover-square, W. (C. 1882-4.)
1893 CURTIS, HENRY JONES, M.D., University College Hospital, W.C.

1884 DAKIN, W. RADFORD, M.D., B.S., 57, Welbeck-street, W.
1884 DALLAWAY, DENNIS, 5, Duchess-street, W.
1883 DALTON, NORMAN, M.D., 4, Mansfield-street, W.
1873 DAVIDSON, ALEXANDER, M.D., 2, Gambier-terrace, Liverpool.
1885 DAVIES, ARTHUR, M.D., 23, Finsbury-square, E.C.
L1869 DAVIES-COLLEY, J. NEVILLE C., M.B., 36, Harley-street, W. (C. 1880-2. V.-P. 1890-1.)
1883 DAVIS, EDWIN HARRY, West Hartlepool.
‡1859 DAVIS, FRANCIS WILLIAM, R.N., 59, Warwick-gardens, W.
1879 DAVY, HENRY, M.D., 29, Southernhay, Exeter.
1866 DAY, WILLIAM HENRY, M.D., 10, Manchester-square, W.
1889 DEAN, HENRY PERCY, M.B., B.S., 84, Wimpole-street, W.
1887 DELÉPINE, SHERIDAN, M.B., 258, Oxford-road, Manchester.
1880 DENT, CLINTON T., 61, Brook-street, W.
1856 DICK, H., M.D.

Elected

1871 DICKINSON, EDWARD HARRIMAN, M.A., M.D., 162, Bedford-street, Liverpool.

1858 DICKINSON, WILLIAM HOWSHIP, M.D., 9, Chesterfield-street, W. (C. 1866-8. S. 1869-71. V.-P. 1872-4. P. 1889-90.)

L1890 DICKINSON, WILLIAM LEE, M.B., 9, Chesterfield-street, W.

1872 DIVER, EBENEZER, M.D., Kenley, Caterham-valley, Surrey.

1872 DORAN, ALBAN HENRY GRIFFITHS, 9, Granville-place, W. (C. 1882-4.)

L1866 DOWN, JOHN LANGDON H., M.D., 81, Harley-street, W. (C. 1872-4.)

1877 DRAKE-BROCKMAN, E. F., Madras Medical Service [care of Mr. Lewis, Gower-street, W.C.].

1880 DRESCHFELD, JULIUS, M.D., 325, Oxford-road, Manchester.

1879 DREWITT, F. G. DAWTREY, M.D., 2, Manchester-square, W. (C. 1890-2.)

L1865 DUCKWORTH, Sir DYCE, M.D., LL.D., 11, Grafton-street, Bond-street, W. (C. 1877.)

L1847 DUDGEON, ROBERT E., M.D., 53, Montagu-square, W.

1865 DUFFIN, ALFRED BAYNARD, M.D., 18, Devonshire-street, Portland-place, W. (C. 1872-4.)

1868 DUKE, OLIVER THOMAS, M.B., India.

1871 DUKES, CLEMENT, M.D., B.S., Sunnyside, Rugby.

1877 DUNBAR, J. J. MACWHIRTER, M.D., Hedingham House, Clapham-common, S.W.

1877 DUNCAN, ANDREW, M.D., 8, Henrietta-street, Covent-garden, W.C.

1889 DUNCAN, JOHN, M.D., St. Petersburg.

1884 DUNN, LOUIS ALBERT, M.B., M.S., 10, St. Thomas's-street, S.E.

1858 DURHAM, ARTHUR EDWARD, 82, Brook-street, W. (C. 1869-71. V.P. 1883-5.)

1879 DURHAM, FREDERIC, M.B., 82, Brook-street, W.

1892 EDDOWES, ALFRED, M.D., 25, Old Burlington-street, W.

L1880 EDMUNDS, WALTER, M.C., 75, Lambeth Palace-road, S.E. (C. 1892-3.)

1882 EDWARDS, F. SWINFORD, 55, Harley-street, W.

1889 ELAM, WILLIAM HENRY, New Barnet, Herts.

1883 ELDER, GEORGE, M.D., 17, Regent-street, Nottingham.

1867 ELLIS, JAMES, M.D., California.

1873 ENGELMANN, GEORGE JULIUS, M.D., A.M., 3003, Locust-street, St. Louis, Miss., U.S.

L1846 ERICHSEN, JOHN ERIC, LL.D., F.R.S., 6, Cavendish-place, W. (C. 1849-51. V.-P. 1863-4.)

1875 EVANS, JULIAN, A.M., M.D., 123, Finborough-road, Redcliffe-square, West Brompton, S.W.

1879 EVE, FREDERIC S., 125, Harley-street, W. (C. 1885-7.)

1876 EWART, JAMES COSSAR, M.B., C.M., F.R.S., School of Medicine, Edinburgh.

1881 EWART, JOSEPH, M.D., Montpellier Terrace, Brighton.

L1877 EWART, WILLIAM, M.D., 33, Curzon-street, W. (C. 1889-1.)

Elected

‡1859 EWENS, JOHN, 14, Whiteladies-road, Clifton, Bristol.

1887 EYLES, CHARLES HENRY, Gold Coast Colony.

1889 FAIRBANK, FREDERICK ROYSTON, M.D., 59, Warrior-square, St. Leonard's-on-Sea.

1872 FAYRER, Sir JOSEPH, K.C.S.I., M.D. LL.D., F.R.S., 53, Wimpole-street, W. (C. 1880-2. V.-P. 1890-1.)

1872 FENN, EDWARD L., M.B., Grey Friars, Colchester.

1883 FENWICK, E. HURRY, 5, Old Burlington-street, W.

1872 FENWICK, JOHN C. J., M.D., 25, North-road, Durham.

L1863 FENWICK, SAMUEL, M.D., 29, Harley-street, W.

1892 FENWICK, W. SOLTAU, M.D., 26, Upper Berkeley-street, W.

1885 FÉRÉ, CHARLES, M.D., Médecin de Bicêtre; Boulevard St. Michel, 37, Paris.

1876 FINLAY, DAVID W., M.D., 2, Queen's-terrace, Aberdeen. (C. 1886-8.)

1859 FISHER, ALEXANDER, M.D., 2, Bruntsfield Gardens, Edinburgh.

1882 FLEMING, GEORGE, C.B., LL.D., Higher Leigh, Combe Martin, North Devon.

1893 FLETCHER, H. MORLEY, M.A., M.B., B.C., 98, Harley-street, W.

1872 FORBES, DANIEL MACKAY, Shoreditch Infirmary, 204, Hoxton-street, N.

‡1866 FOSTER, Sir BALTHAZAR WALTER, M.D., M.P., 11, George-street, Hanover-square, W.

1872 FOTHERBY, HENRY J., M.D., Woodthorpe Cotes, Reigate.

1891 FOULERTON, ALEXANDER GRANT RUSSELL, 122, Brompton-road, S.W.

1880 FOWLER, JAMES KINGSTON, M.A., M.D., 35, Clarges-street, W. (C. 1887-8.)

1878 FOX, THOMAS COLCOTT, M.B., B.A., 14, Harley-street, W. (C. 1892-3.)

1858 FRANCIS, CHARLES RICHARD, M.B., Bengal Medical Establishment, Indian Army.

1892 FREDERICK, HERBERT J., St. Thomas's Hospital, S.E.

1886 FREEMAN, HENRY WILLIAM, 24, Circus, Bath.

O.M. FRERE, J. C.

1891 FRIPP, ALFRED DOWNING, M.B., B.S., 65, Harley-street, W.

1864 FRODSHAM, JOHN MILL, M.D., Streatham, S.W.

1880 GABBETT, HENRY SINGER, M.B., 20, Burlington-place, Eastbourne.

‡1858 GAIRDNER, WILLIAM TENNANT, M.D., LL.D.Edin., F.R.S., 225, St. Vincent-street, Glasgow. (V.-P. 1891-2.)

1890 GALLOWAY, JAMES, M.A., M.B., 16, Finsbury-circus, E.C.

1870 GALTON, JOHN H., M.D, 39, Anerley-road, Upper Norwood, S.E.

1855 GAMGEE, J.

L1846 GARROD, Sir ALFRED BARING, M.D., F.R.S., 10, Harley-street, W. (C. 1851. V.-P. 1863-5.)

1892 GARROD, ARCHIBALD EDWARD, M.D., 9, Chandos-street, W.

1879 GARSTANG, THOMAS WALTER HARROPP, Headingley House, Knutsford, Cheshire.

Elected

1872 Garton, William, M.D., Inglewood, Aughton, near Ormskirk, Lancashire.

1891 Gaster, Aughel, M.D., 34, Warwick-road, Maida-hill, W.

1880 Gibbes, Heneage, M.B., University of Michigan, Ann Arbor, Michigan, U.S.A.

L1853 Gibbon, Septimus, M.D., 39, Oxford-terrace, Hyde-park, W.

1878 Gibbons, R. A., M.D., 29, Cadogan-place, S.W.

1876 Gill, John, M.D., 31, Apsley-road, Clifton, Bristol.

1881 Glynn, Thomas Robinson, M.D., 62, Rodney-street, Liverpool.

1873 Godlee, Rickman John, M.B., M.S., 19, Wimpole-street, W. (C. 1877-80, 1891-2. S. 1887-9. V.-P. 1893.)

1878 Golding-Bird, Cuthbert H., M.B., 12, Queen Anne-street, W. (C. 1885-7.)

1890 Goodall, E. Wilberforce, M.D., The Eastern Hospital, Homerton, N.E.

1871 Goodhart, James Frederic, M.D., 25, Weymouth-street, W. (C. 1876-8, 1886-8. S. 1883-5. V.-P. 1892-3.)

1875 Gould, Alfred Pearce, M.S., 10, Queen Anne-street, W. (C. 1883-5.)

1870 Gowers, William Richard, M.D., F.R.S., 50, Queen Anne-street, W. (C. 1878-9.)

L1858 Gowlland, Peter Y., 82, Gloucester-terrace, Hyde-park, W.

1888 Grant, J. Dundas, M.A., M.D., C.M., 8, Upper Wimpole-street, W.

1867 Green, T. Henry, M.D., 74, Wimpole-street, W. (C. 1871-3, 1878-9. S. 1875-6. V.-P. 1886-8.)

1873 Greenfield, William Smith, M.D., B.S., 7, Heriot-row, Edinburgh. (C. 1877-80. V.-P. 1893.)

‡1855 Greenhill, William Alexander, M.D., 5, The Croft, Hastings.

1886 Greves, Edwin Hyla, M.D., Rodney House, Suffolk-road, Bournemouth.

1885 Griffith, Walter Spencer Anderson, M.B., 114, Harley-street, W.

1892 Griffith, William Stokes, M.B., B.C., 4, Bramham-gardens, S.W.

1887 Griffiths, Joseph, M.B., C.M., 16, Panton-street, Cambridge.

1876 Griffiths, Thomas D., M.D., Hearne Lodge, Swansea.

1882 Gross, Charles, M.D., M.S., 112, Westbourne-grove, W.

1880 Gunn, R. Marcus, M.B., C.M., 54, Queen Anne-street, W.

1876 Gwyther, James, M.B.

1887 Habershon, Samuel Herbert, M.D., 70, Brook-street, W.

L1851 Hacon, E. Dennis, 269, Mare-street, Hackney, N.E. (C. 1872.)

1892 Hadley, Wilfred James, M.D., 16, Wimpole-street, W.

1882 Haig, A., M.B., 7, Brook-street, W.

1877 Hallowes, Frederick Blackwood, Redhill, Surrey.

1890 Hamer, William Heaton, M.B., Ladywell, Dartmouth Park Hill, Highgate, N.

1886 Hamilton, David James, M.B., 1, Albyn-place, Aberdeen.

1890 Handfield-Jones, Montagu, M.D., 35, Cavendish-square, W.

Elected

1886 HANDFORD, HENRY, M.D., 14, Regent-street, Nottingham.
1891 HANKIN, E. H., St. John's College, Cambridge.
1882 HARBINSON, ALEXANDER, M.D., County Lunatic Asylum, Lancaster.
L1848 HARE, CHARLES JOHN, M.D., Berkeley House, 15, Manchester-square, W. (C. 1852-4. V.-P.1874-7.)
L1856 HARLEY, GEORGE, M.D., F.R.S., 25, Harley-street, W. (C. 1862-5. V.-P. 1878-80.)
1872 HARRIS, HENRY, M.D., Trengweath-place, Redruth, Cornwall.
1879 HARRIS, VINCENT DORMER, M.D., 31, Wimpole-street, W.
L1858 HART, ERNEST, 38, Wimpole-street, W. (C. 1867-8.)
1891 HASLAM, WILLIAM F., 33, Paradise-street, Birmingham.
1870 HAWARD, JOHN WARRINGTON, 16, Savile-row, W. (C. 1879-81. V.-P. 1890-1.)
1886 HAWKINS, FRANCIS HENRY, M.B., 59, Wimpole-street, W.
1890 HAWKINS, HERBERT PENNELL, M.B., 109, Harley-street, W.
L1856 HEATH, CHRISTOPHER, 36, Cavendish-square, W. (C. 1866-7. V.-P. 1879-81.)
1892 HEATON, GEORGE, M.B., 33, Temple-row, Birmingham.
1881 HEBB, RICHARD G., M.D., 9, Suffolk-street, S.W. (C. 1891-3.)
1884 HEBBERT, CHARLES ALFRED.
1878 HELLIER, JOHN B., M.B., Headingley, Leeds.
1879 HENDERSON, GEORGE COURTENAY, M.D., Kingston, Jamaica, West Indies.
1892 HENRY, ROBERT, Moorcroft, Hillingdon, Uxbridge.
1869 HENSLEY, PHILIP J., M.D., 4, Henrietta-street, W.
1884 HERRINGHAM, WILMOT PARKER, M.B., 13, Upper Wimpole-street, W.
1892 HEWLETT, RICHARD TANNER, M.B., King's College, Strand, W.C.
1864 HICKMAN, WILLIAM, M.B., 5, Harley-street, W. (C. 1890-2.)
1880 HOBSON, JOHN MORRISON, M.D., Glendalough, Morland-road, Croydon.
L1854 HOLMES, TIMOTHY, 18, Great Cumberland-place, W. (C. 1862-3. S. 1864-7. C. 1868. V.-P. 1869-71.)
L1850 HOLT, BARNARD WIGHT, 14, Savile-row, W. (C. 1853.)
O.M. HOLTHOUSE, CARSTEN. (C. 1852-4, V.-P. 1874-5.)
1878 HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green-street,
1864 HOOD, WHARTON P., M.D., 11, Seymour-street, W.
1870 HOPE, WILLIAM, M.D., 54, Curzon-street, W.
1882 HOPKINS, JOHN, Central London Sick Asylum, Cleveland-street, W.
1879 HORROCKS, PETER, M.D., 26, St. Thomas's-street, S.E.
1883 HORSLEY, VICTOR, M.B., B.S., F.R.S., 25, Cavendish-square, W. (C. 1888-9.)
L1880 HOVELL, T. MARK, 105, Harley-street, W.
1875 HOWSE, HENRY GREENWAY, M.S., 59, Brook-street, W. (C. 1878-81.)
1884 HUDSON, CHARLES ELLIOTT LEOPOLD BARTON 6, Chandos-street, W.
L1856 HUDSON, JOHN, M.D., 11, Cork-street, W.
L1854 HULKE, JOHN WHITAKER, F.R.S., 10, Old Burlington-street, W. (C. 1863-5. S. 1868-72. V.-P. 1873-6, 1885-6. T. 1877-9. P. 1883-4.)

Elected

1874 HUMPHREYS, HENRY, M.D., Victoria-road, Fleet, Hants.

1883 HUMPHRY, Sir GEORGE MURRAY, M.D., LL.D., F.R.S., University of Cambridge. (P. 1891-2.)

1888 HUNTER, WILLIAM, M.D., 54, Harley-street, W.

1852 HUTCHINSON, JONATHAN, F.R.S., 15, Cavendish-square, W. (C. 1856-9. V.-P. 1872-3, 1881-3. P. 1879-80.)

1882 HUTCHINSON, JONATHAN, jun., 1, Park-crescent, W. (C. 1889-91.)

1884 HUTTON, HENRY RICHMOND, M.B., 8A, St. John-street, Manchester.

1889 HYSLOP, THEOPHILUS BULKELEY, M.B., C.M., Bethlem Royal Hospital, S.E.

1880 INGRAM, ERNEST FORTESCUE, Newcastle, Natal, S. Africa.

1886 JACKSON, ARTHUR MOLYNEUX, M.D., Kent County Asylum, Barming Heath, Maidstone.

1865 JACKSON, J. HUGHLINGS, M.D., F.R.S., 3, Manchester-square, W. (C. 1872-3. V.-P. 1888-9.)

1886 JACKSON, PHILIP J., 216, Great Dover-street, S.E.

1887 JACOB, ERNEST HENRY, M.D., 12, Park-street, Leeds.

1875 JALLAND, WILLIAM HAMERTON, St. Leonard's House, Museum-street, York.

1888 JAMES, JAMES THOMAS, M.D., 30, Harley-street, W.

‡1853 JARDINE, JOHN LEE, Capel, near Dorking, Surrey.

1881 JENNINGS, WILLIAM OSCAR, M.D., 8, Rue Roy, Paris.

1879 JESSOP, CHARLES MOORE, 98, Sutherland-avenue, W.

1866 JESSOP, THOMAS RICHARD, 31, Park-square, Leeds.

1878 JOHNSON, ARTHUR JUKES, Yorkville, Ontario, Canada.

1876 JOHNSON, CHARLES HENRY, Winton House, Basingstoke, Hants.

L.O.M. JOHNSON, Sir GEORGE, M.D., F.R.S., 11, Savile-row, W. (C. 1846-50. V.-P. 1863-4, 1884-6. T. 1880-3.)

1888 JOHNSON, RAYMOND, M.B., B.S., 20, Weymouth-street, Portland-place, W.

1881 JOHNSTON, JOSEPH, M.D., 24, St. John's Wood-park, N.W.

1854 JOHNSTONE, ATHOL A. W., St. Moritz House, 61, Dyke-road, Brighton.

L1853 JONES, SYDNEY, M.B., 16, George-street, Hanover-square, W. (C. 1864-6. V.-P. 1886-7.)

1888 JONES, TALFOURD, M.B., Eastbourne.

1862 JONES, THOMAS RIDGE, M.D., 4, Chesham-place, S.W. (C. 1882-4.)

1886 JULER, HENRY EDWARD, 23, Cavendish-square, W.

1890 KANTHACK, A. A., M.B., B.S., 31, Rodney-street, Liverpool.

1867 KELLY, CHARLES, M.D., Broadwater-road, Worthing, Sussex. (C. 1874.)

L1846 KENT, THOMAS J., 89, Piccadilly, W.

1879 KESTEVEN, WILLIAM HENRY, Hillwood, Waverley-grove, Hendon, N.W.

1859 KIALLMARK, HENRY WALTER, 5, Pembridge-gardens, W. (C. 1875-6.)

1882 KIDD, PERCY, M.D., 60, Brook-street, W. (C. 1889-91.)

1867 KING, EDWIN HOLBOROW, Netley Court, Southampton.

Elected

1871 KING, ROBERT, M.B., Boyfield House, Moulton, Spalding, Lincolnshire.

L1852 KINGDON, J. ABERNETHY, 2, Bank-buildings, E.C.

1888 KYNSEY, WILLIAM RAYMOND, C.M.G., Colombo, Ceylon.

1878 LANCEREAUX, ETIENNE, M.D., 44, Rue de la Bienfaisance, Paris.

1882 LANE, WILLIAM ARBUTHNOT, M.B., M.S., 8, St. Thomas's-street, S.E. (C. 1891-3.)

1865 LANGTON, JOHN, 62, Harley-street, W. (C. 1882-4.)

1886 LANKESTER, HERBERT, M.D., 1, Elm-park-gardens, S.W.

1869 LARCHER, O., M.D.Par., 97, Rue de Passy, Paris. [M. Kliensieck, Libraire, Rue de Lille, 11, Paris, per Messrs. Longmans.]

1884 LARDER, HERBERT, Whitechapel Infirmary, Bakers-row, E.

1873 LATHAM, PETER WALLWORK, M.D., 17, Trumpington-street, Cambridge.

1876 LAW, WILLIAM THOMAS, M.D., 9, Norfolk-crescent, W.

1883 LAWFORD, JOHN BOWRING, M.D., C.M., 55, Queen Anne-street, W.

1853 LAWRENCE, HENRY JOHN HUGHES, Picton House, Llandowror, St. Clears. (C. 1873-5.)

1892 LAWRENCE, TURNER WILLIAM PELHAM, M.B., 138, Maida-vale, W.

1893 LAWSON, ARNOLD, 12, Harley-street, W.

1859 LAWSON, GEORGE, 12, Harley-street, W. (C. 1870-1. V.-P. 1884-5.)

1879 LAYCOCK, GEORGE LOCKWOOD, M.B., Melbourne, Victoria, Australia.

1891 LAZARUS-BARLOW, WALTER, M.B.(Sydney), The Acacias, Chesterton, Cambridge.

1875 LEDIARD, HENRY AMBROSE, M.D., 41, Lowther-street, Carlisle.

L1852 LEE, HENRY, 9, Savile-row, W. (C. 1860-2. V.-P. 1875-6.)

1879 LEECH, DANIEL JOHN, M.D., 96, Mosley-street, Manchester.

L1877 LEES, DAVID B., M.D., 22, Weymouth-street. W. (C. 1890-2.)

1867 LEES, JOSEPH, M.D., 21, Brixton-road, S.W.

1877 LEESON, JOHN RUDD, M.D., C.M., 6, Clifden-road, Twickenham.

1868 LEGG, JOHN WICKHAM, M.D. (C. 1874-5.)

1892 LEITH, ROBERT FRASER CALDIE, M.B., C.M., B.Sc., 129, Warrender-park-road, Edinburgh.

1892 LEUDET, THEODORE-EMILE, 49, Boulevard Cauchoise, Rouen, France.

1861 LICHTENBERG, GEORGE, M.D., 47, Finsbury-square, E.C.

1889 LIGHT, EDWIN MELLOR, M.B., B.C., The Infirmary, Leeds.

1877 LISTER, Sir JOSEPH, Bart., D.C.L., LL.D., F.R.S., 12, Park-crescent, W. (C. 1880-2. V.-P. 1887-8, 1891-2.)

1889 LITTLE, JOHN FLETCHER, M.B., 32, Harley-street, W.

L1862 LITTLE, LOUIS S., China.

1878 LITTLEJOHN, SALTER G., M.B., C.M., Central London District Schools, Hanwell.

1874 LIVEING, EDWARD, M.D., 52, Queen Anne-street, W.

L1863 LIVEING, ROBERT, M.D., 11, Manchester-square, W. (C. 1876.)

1882 LOCKWOOD, C. B., 19, Upper Berkeley-street, W. (C. 1893.)

Elected

1876 LONGHURST, ARTHUR EDWIN TEMPLE, M.D., 4, Eaton Square, S.W. (C. 1885-7.)
1892 LOVELL, C., Wimbledon.
1881 LUBBOCK, MONTAGU, M.D., 19, Grosvenor-street, W.
1873 LUCAS, R. CLEMENT, M.B., M.S., 18, Finsbury-square, E.C. (C. 1883-5.)
1880 LUND, EDWARD, 23, St. John-street, Manchester.
1879 LUNN, JOHN REUBEN, St. Marylebone Infirmary; Rackham-street, Ladbroke-grove-road, W.
1887 LYON, THOMAS GLOVER, M.D., 8, Finsbury-circus, E.C.

1871 MACCARTHY, JEREMIAH, M.A., 15, Finsbury-square, E.C. (C. 1878-80.)
1873 MCCONNELL, J. F.
1871 MAC CORMAC, Sir WILLIAM, 13, Harley-street, W. (C. 1878-80.)
1882 MACKENZIE, FREDERIC MORELL, 29, Hans-place, S.W.
1870 MACKENZIE, GEORGE WELLAND, 13, William-street, S.W.
1885 MACKENZIE, HECTOR WILLIAM GAVIN, M.A., M.D., 59, Welbeck-street, W.
1870 MACKENZIE, JOHN T., Bombay, India.
1878 MACKENZIE, STEPHEN, M.D., 18, Cavendish-square, W. (C. 1888-90.)
1879 MACLAGAN, THOMAS JOHN, M.D., 9, Cadogan-place, S.W.
1865 MACLAURIN, HENRY NORMAND, M.D., 187, Macquarie-street, Sydney, New South Wales.
1876 MACNAMARA, CHARLES, 13, Grosvenor-street, W.
1879 MACREADY, JONATHAN FORSTER, 51, Queen Anne-street, W.
1885 MAGUIRE, ROBERT, M.D., 4, Seymour-street, W.
1877 MAKINS, GEORGE HENRY, 47, Charles-street, Berkeley-square, W. (C. 1889-91.)
1887 MALCOLM, JOHN DAVID, M.B., C.M., 13, Portman-street, W.
1892 MANN, HAROLD EDWARD, Alderney.
1890 MANSON, PATRICK, M.D., C.M., 21, Queen Anne-street, W.
1876 MAPLES, REGINALD, Kingsclere, near Newbury.
1868 MARSH, F. HOWARD, 30, Bruton-street, W. (C. 1876-7.) (V.-P. 1889-90.)
1887 MARTIN, SIDNEY, M.D., B.S., 10, Mansfield-street, W. (C. 1893.)
1889 MASON, DAVID JAMES, M.D., Rosemont, Maidenhead.
1867 MASON, PHILIP BROOKES, Burton-on-Trent.
1892 MASTERS, JOHN ALFRED, M.D., 35, Bruton-street, W.
1884 MAUDSLEY, HENRY CARR, M.D., 11, Spring-street, Melbourne, Victoria.
L1852 MAY, GEORGE, jun., M.B., Reading.
1888 MAY, WILLIAM PAGE, M.D., B.Sc., care of the Countess Visconti, Puttenham Priory, Guildford.
1881 MAYLARD, ALFRED ERNEST, M.B., 4, Berkeley-terrace, Glasgow.
1874 MEREDITH, WILLIAM APPLETON, M.B., 21, Manchester-square, W.
1859 MESSER, JOHN COCKBURN, M.D., Assistant Surgeon R.N.
1866 MICKLEY, GEORGE, M.A., M.B., St. Luke's Hospital, Old-street, E.C.

Elected

1882 Money, Angel, M.D.
1879 Moore, Norman, M.D., 94, Gloucester-place, Portman-square, W. (C. 1885-7.)
1881 Moore, Thomas, 6, Lee-terrace, Blackheath, S.E.
1875 Morgan, John H., 68, Grosvenor-street, W. (C. 1886-8.)
1874 Morison, Alexander, M.D., C.M., Dunnottar, 115, Green-lanes, N.
1880 Morison, Basil Gordon, M.B., C.M., 70, Marquess-road, N.
1869 Morris, Henry, M.A., M.B., 8, Cavendish-square, W. (C. 1877-9, 1884-6. S. 1881-3. V.-P. 1888-9.)
1879 Morris, Malcolm Alexander, 8, Harley-street, W.
1891 Morton, Charles A., 24, St. Paul's-road, Clifton, Bristol.
1875 Morton, John, M.B., Guildford.
1884 Mott, Frederick Walker, M.D., 84, Wimpole-street, W. (C. 1891-3.)
1879 Moullin, Charles W. Mansell, M.D., 69, Wimpole-street, W.
1878 Mumford, William Lugar, M.D., 12, Suffolk-street, Pall Mall, S.W.
1893 Mummery, John Howard, M.R.C.S., L.D.S.Eng., 10, Cavendish-place, W.
1876 Munro, William, M.D., C.M., Clarkefield, Fountain-street, Cheetham Hill, Manchester.
1885 Murray, Hubert Montague, M.D., 27, Savile-row, W.
1864 Myers, Arthur B. R., 43, Gloucester-street, Warwick-square, S.W. (C. 1872-3.)
1882 Myers, A. T., M.D., 2, Manchester-square, W.
1887 Myers, W. H., Fort Wayne, Indiana, U.S.A.

1887 Nason, Edward Noel, M.B., 80, Abbey-street, Nuneaton.
1873 Nettleship, Edward, 5, Wimpole-street, W. (C. 1882-4.)
1875 Newby, Charles Henry, 15, Landport-terrace, Southsea, Hants.
1865 Newman, William, M.D., Stamford, Lincolnshire.
1868 Nicholls, James, M.D., Trenarren, Newquay, Cornwall.
1876 Nicholson, John Francis, M.D., 29, Albion-street, Hull.
1878 Noott, William Mathias, 8, Kensington-park-road, W.
L1864 Norton, Arthur T., 101, Harley-street, W. (C. 1877-9.)
1883 Norvill, Frederic Harvey, M.B., Summerland, Yeovil, Somersetshire.
1856 Nunn, Thomas William, 8, Stratford-place, W. (C. 1864-6. V.-P. 1878-80.)
1871 Nunneley, Rev. Frederick Barham, M.D.

1880 O'Connor, Bernard, M.D., Greenhill-park, Harlesden, N.W.
1873 O'Farrell, George Plunkett, M.B., 19, Fitzwilliam-square, Dublin.
1880 Ogilvie, George, M.B., 22, Welbeck-street, W.
1880 Ogilvie, Leslie, M.B., 46, Welbeck-street, W.
1850 Ogle, John W., M.D., 30, Cavendish-square, W. (C. 1855-6. S. 1857-60. C. 1861-3. V.-P. 1865-8.)
1876 Oliver, John Ferens, M.D., 12, Old Elvet, Durham.
1888 Openshaw, Thomas Horrocks, M.B., B.S., 16, Wimpole-street, W.
1860 Orange, William, M.D., 12, Lexham-gardens, W.

Elected

1875 ORD, WILLIAM MILLER, M.D., 37, Upper Brook-street, W. (C. 1880-2. V.-P. 1893.)

1892 ORD, WILLIAM WALLIS, M.B., B.A., 2, Queen-street, Mayfair, W.

1879 ORMEROD, J. A., M.D., 25, Upper Wimpole-street, W. (C. 1887-9.)

1881 OWEN, ISAMBARD, M.D., 40, Curzon-street, W.

1865 OWLES, JAMES ALLDEN, M.D., Hill View, Woking, Surrey.

1875 PAGE, HERBERT WILLIAM, M.A., M.C. Cantab., 146, Harley-street, W. (C. 1889-91.)

1870 PAGET, Sir JAMES, Bart., D.C.L., LL.D., F.R.S., 5, Park-place, W. (P. 1887-8.)

1884 PAGET, STEPHEN, 57, Wimpole-street, W.

1872 PARKER, ROBERT WILLIAM, 13, Welbeck-street, W. (C. 1881-3.)

1874 PARKER, RUSHTON, M.B., B.S., 59, Rodney-street, Liverpool.

L1853 PARKINSON, GEORGE, 50, Brook-street, W.

1882 PASTEUR, WILLIAM, M.D., 4, Chandos-street, W. (C. 1893.)

1885 PAUL, FRANK THOMAS, 44, Rodney-street, Liverpool.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S. (PRESIDENT), 35, Grosvenor-street, W. (C. 1872-4. V.-P. 1891-2. P. 1893.)

1868 PAYNE, JOSEPH FRANK, M.D., 78, Wimpole-street, W. (C. 1873-5, 1883-5. S. 1880-2. V.-P. 1888-9.)

1872 PEARCE, JOSEPH CHANING, M.D., C.M., Montague House, St. Lawrence-on-Sea, Kent.

1863 PEARSON, DAVID R., M.D., 23, Upper Phillimore-place, W.

1884 PEDLEY, F. NEWLAND, 32, Devonshire-place, Portland-place, W.

1879 PEEL, ROBERT, 130, Collins-street East, Melbourne, Victoria.

1889 PENBERTHY, JOHN, Royal Veterinary College, Camden Town, N.W.

1887 PENROSE, FRANCIS GEORGE, M.D., 4, Harley-street, W.

1884 PEPPER, AUGUSTUS JOSEPH, M.B., C.M., 13, Wimpole-street, W.

1888 PERRY, EDWIN COOPER, M.B., The College, Guy's Hospital, S.E.

1878 PHILIPPS, SUTHERLAND REES, M.D., St. Ann's-heath, Virginia Water, Chertsey.

1871 PHILLIPS, CHARLES DOUGLAS F., M.D., 10, Henrietta-street, W.

1878 PHILLIPS, JOHN WALTER, 30, Stanley-street, West Melbourne, Victoria.

1863 PICK, THOMAS PICKERING, 18, Portman-street, W. (C. 1870-1. V.-P. 1885-7.)

1893 PINKERTON, ROBERT A., M.A., M.D., Surrey Dispensary, Great Dover-Street, S.E.

1867 PITT, EDWARD G., M.D.

1884 PITT, GEORGE NEWTON, M.D., 24, St. Thomas's-street, S.E. (C. 1890-2.)

1876 PITTS, BERNARD, M.A., M.B., 109, Harley-street, W. (C. 1888-90.)

1887 PITTS, ROBERT ZACCHEUS, Springfield, Chelmsford.

1883 POLAND, JOHN, 4, St. Thomas's-street, Southwark, S.E.

1882 POLLARD, BILTON, 24, Harley-street, W.

L1846 POLLOCK, GEORGE D. (TRUSTEE), 36, Grosvenor-street, W. (S. 1850-3. C. 1854-6. V.-P. 1863-5. P. 1875-6.)

Elected

1850 POLLOCK, JAMES EDWARD, M.D., 52, Upper Brook-street, W. (C. 1862-4. V.-P. 1879-81.)

1870 POORE, GEORGE VIVIAN, M.D., 30, Wimpole-street, W. (C. 1883-5.)

1876 PORT, HEINRICH, M.D., 48, Finsbury-square, E.C.

L1879 POTTER, HENRY PERCY, St. Mary Abbotts' Infirmary, Marloes-road, Kensington, W.

1866 POWELL, RICHARD DOUGLAS, M.D., 62, Wimpole-street, W. (C. 1873-5, 1881-3. S. 1877-9. V.-P. 1887-8.)

1884 POWER, D'ARCY, M.A., M.B., 26, Bloomsbury-square, W.C. (C. 1891-3.)

1865 POWER, HENRY, 37A, Great Cumberland-place, W. (C. 1876-7.)

1887 PRATT, WILLIAM SUTTON, Weedon, Northamptonshire.

1884 PRICE, J. A. P., M.D., 41, Castle-street, Reading.

L1856 PRIESTLEY, Sir WILLIAM OVEREND, M.D., 17, Hertford-street, W.

1888 PRIMROSE, ALEXANDER, M.B., C.M. 196, Simcoe-street, Toronto, Canada.

1882 PRINGLE, J. J., M.B., 23, Lower Seymour-street, W.

L1848 PURNELL, JOHN JAMES, Woodlands, Streatham-hill, S.W. (C. 1858-61.)

1865 PYE-SMITH, PHILIP HENRY, M.D., F.R.S., 48, Brook-street, W. (C. 1874-7. V.-P. 1890-1.)

LO.M. QUAIN, Sir RICHARD, Bart., M.D., LL.D., F.R.S. (TRUSTEE), 67, Harley-street, W. (C. 1846-51. S. 1852-6. T. 1857-68. *Pres.* 1869-70. V.-P. 1871-3.)

1884 RAKE, BEAVEN NEAVE, M.D., Leper Asylum, Trinidad.

1872 RALFE, CHARLES HENRY, M.D., M.A., 26, Queen Anne-street, W. (C. 1877-9.)

1857 RANKE, HENRY, M.D., Munich.

‡1890 RANSOM, WILLIAM BRAMWELL, M.D., The Pavement, Nottingham.

1865 RASCH, ADOLPHUS A., M.D., 7, South-street, Finsbury-square, E.C.

1891 RATCLIFFE, JOSEPH RILEY, M.B., The General Hospital, Birmingham.

1887 RAVEN, THOMAS FRANCIS, Broadstairs, Kent.

1870 RAY, EDWARD REYNOLDS, Dulwich Village, S.E.

1875 REID, ROBERT WILLIAM, M.D., C.M., 8, Queen's-gardens, Aberdeen.

1881 RENNER, WILLIAM, M.R.C.S., Wilberforce-street, Free Town, Sierra Leone.

1854 REYNOLDS, J. RUSSELL. M.D., F.R.S., 38, Grosvenor-street, W. (C. 1868-9.)

1871 RICHARDS, J. PEEKE, 6, Freeland-road, Ealing, W.

1866 RIVINGTON, WALTER, M.S., 95, Wimpole-street, W.

‡1865 ROBERTS, DAVID LLOYD, M.D., 11, St. John's-street, Manchester.

1871 ROBERTS, FREDERICK THOMAS, M.D., 102, Harley-street, W. (C. 1883-5.)

1878 ROBERTS, WILLIAM HOWLAND, M.D., Surgeon, Madras Army.

1888 ROBERTSON, ROBERT, M.D., The Bungalow, Ventnor, Isle of Wight.

1885 ROBINSON, ARTHUR HENRY, M.D., Mile End Infirmary, Bancroft-road, N.E.

1887 ROBINSON, HENRY BETHAM, M.B., 6, Mansfield-street, W.

1882 ROBINSON, TOM, M.D., 9, Princes-street, Cavendish-Square, W.

Elected

1888 Rolleston, H. D., M.D., M.A., 13, Upper Wimpole-street, W.

L1858 Rose, Henry Cooper, M.D., Penrose House, Hampstead, N.W. (C. 1873-4.)

1876 Rose, William, M.B., B.S., 17, Harley-st., W.

1875 Rossiter, George Frederick, Cairo Lodge, Weston-super-Mare.

L1877 Roth, Bernard, 29, Queen Anne-street.

1888 Roughton, Edmund Wilkinson, 33, Westbourne-terrace, W.

1891 Rouillard, Laurent Antoine John, M.B., St. Thomas's Hospital, S.E.

1858 Rouse, James, 2, Wilton-street, S.W.

1887 Roy, Charles Smart, F.R.S., M.D., M.A., University of Cambridge.

1891 Ruffer, Marc Armand, M.D., 5, York-terrace, Regent's-park, N.W.

1891 Russell, William, M.D., 46, Albany-street, Edinburgh.

1869 Rutherford, William, M.D., F.R.S., 14, Douglas-crescent, Edinburgh.

1882 Sainsbury, Harrington, M.D., 63, Welbeck-street, W.

1853 Salter, S. James A., M.B., F.R.S., Basingfield, near Basingstoke, Hants. (C. 1861-3. V.-P. 1880-2.)

1854 Sanderson, John Burdon, M.D., D.C.L.Durham, F.R.S., 50, Banbury-road, Oxford. (C. 1864-7. V.-P. 1873-4.)

1875 Sangster, Charles, 148, Lambeth-road, S.E.

1877 Sankey, H. R. O., M.B., Boreatton Park, Baschurch, Salop.

1886 Saundby, Robert, M.D., 83A, Edmund-street, Birmingham.

1871 Saunders, Charles Edward, M.D., Sussex County Lunatic Asylum, Hayward's Heath.

1890 Saunders, Frederick William, M.B., B.C., 17, Barkston-gardens, South Kensington, S.W.

1873 Savage, George Henry, M.D., 3, Henrietta-street, Cavendish-square, W. (C. 1881-3.)

1882 Savill, Thomas Dixon, M.D., 12, Upper Berkeley-street, W.

1891 Schoebstein, Gustave Isidore, M.B., B.S., 11, Portland-place, W.

1877 Semon, Felix, M.D., 39, Wimpole-street, W. (C. 1885-7.)

1872 Sergeant, Edward, Town Hall, Bolton, Lancashire.

1876 Sharkey, Seymour J., M.D., 2, Portland-place, W. (C. 1884-6.)

1880 Shattock, Samuel G., 4, Crescent-road, The Downs, Wimbledon. (C. 1885-7, 1893. S. 1890-2.)

1885 Shaw, Lauriston Elgie, M.D., 10, St. Thomas's-street, S.E.

1884 Sheild, Arthur Marmaduke, M.B., B.S., 20, Stratford-place, Oxford-street, W.

L1886 Sherrington, Charles Scott, M.B., F.R.S., 27, St. George's-square, S.W.

L1856 Shillitoe, Buxton, 2, Frederick's-place, E.C.

1887 Sibley, Walter Knowsley, 7, Harley-street, W.

1875 Siddall, Joseph Bowes, M.D., C.M., Conybeare, Northam, Bideford.

1880 Silcock, A. Quarry, M.D., B.S., 52, Harley-street, W. (C. 1888-90.)

1892 Silk, John Frederick William, M.D., 29, Weymouth-street, W.

1866 Sims, Francis Manley Boldero, 12, Hertford-street, W.

Elected

1892 SLATER, CHARLES, M.B., 16, Northwick-terrace, N.W.

1887 SMALLPEICE, WILLIAM DONALD, 42, Queen Anne's-gate, S.W.

‡1875 SMEE, ALFRED HUTCHINSON, The Grange, Hackbridge, Carshalton, Surrey.

1879 SMITH, E. NOBLE, 24, Queen Anne-street, W.

1887 SMITH, FREDERICK JOHN, M.B., 4, Christopher-street, Finsbury-square, E.C.

1875 SMITH, GEORGE JOHN MALCOLM, M.B., Hurstpierpoint, Sussex.

1872 SMITH, GILBART, M.D., 68, Harley-street, W.

1878 SMITH, HERBERT URMSON, M.B., Cape Colony.

1873 SMITH, RICHARD T., M.D., 53, Haverstock-hill, N.W.

1883 SMITH, ROBERT PERCY, M.D., Bethlem Royal Hospital, St. George's Road, S.E.

1869 SMITH, ROBERT SHINGLETON, M.D., Deepholm, Clifton Park, Bristol.

1892 SMITH, SOLOMON CHARLES, M.D., 4, Portman Mansions, Baker-street, W.

1856 SMITH, THOMAS, 5, Stratford-place, W. (C. 1867-9. V.-P. 1877-8.)

1866 SMITH, WILLIAM, Melbourne, Australia.

1870 SMITH, WILLIAM JOHNSON, Seamen's Hospital, Greenwich, S.E. (C. 1879-81.)

L1869 SMITH, WILLIAM WILBERFORCE, M.D., 14, Stratford-place, W.

1870 SNOW, WILLIAM VICARY, M.D., Richmond Gardens, Bournemouth.

1888 SOLLY, ERNEST, M.B., Strathlea, Harrogate, Yorks.

1868 SOUTHEY, REGINALD, M.D., 32, Grosvenor-road, Pimlico. (C. 1882-4.)

1887 SPENCER, WALTER GEORGE, M.B., 35, Brook-street, W.

1888 SPICER, ROBERT HENRY SCANES, M.D., 28, Welbeck-street, W.

1861 SQUIRE, ALEXANDER BALMANNO, M.B., 24, Weymouth-street, W.

1885 SQUIRE, JOHN EDWARD, M.D., 53, Harley-street, W.

1890 STABB, EWEN CARTHEW, St. Thomas's Hospital, Albert-embankment, S.E.

1889 STEWART, EDWARD, M.D.

L1854 STEWART, WILLIAM EDWARD, 16, Harley-street, W.

1891 STILES, HAROLD JALLAND, M.B., C.M., 5, Castle-terrace, Edinburgh.

1879 STIRLING, EDWARD CHARLES, Adelaide, South Australia [care of Messrs. Elder & Co., 7, St. Helen's Place, E.C.].

1883 STOKER, GEORGE, 14, Hertford-street, W.

1881 STOKES, HENRY FRASER, 2, Highbury-crescent, N.

1884 STONHAM, CHARLES, 4, Harley-street, W. (C. 1893.)

1875 STURGE, W. A., M.D., 29, Boulevard Dubouchage, Nice.

L1871 SUTHERLAND, HENRY, M.D., 6, Richmond-terrace, Whitehall, S.W.

1882 SUTTON, JOHN BLAND, 48, Queen Anne-street, W. (C. 1887-90.)

‡1867 SWAIN, WILLIAM PAUL, 17, The Crescent, Plymouth.

1851 SYMONDS, CHARTERS JAMES, M.S., 26, Weymouth-street, Portland-place, W. (C. 1886-8.)

1870 TAIT, ROBERT LAWSON, 7, The Crescent, Birmingham.

1886 TARGETT, JAMES HENRY, M.B., M.S., 6, St. Thomas's-street, S.E.

Elected

1891 TATE, WALTER WILLIAM HUNT, 2, The Terrace, Camden-square, N.W.
1864 TATHAM, JOHN, 12, George-street, Hanover-square, W.
1870 TAY, WARREN, 4, Finsbury-square, E.C. (C. 1881-2.)
1871 TAYLOR, FREDERICK, M.D., 20, Wimpole-street, W. (C. 1879-81.)
1885 TAYLOR, HENRY H., 10, Brunswick-place, Brighton.
1892 TAYLOR, JAMES, M.D.Edin., 45, Weymouth-street, W.
1880 TAYLOR, SEYMOUR, M.D., M.C., 16, Seymour-street, W.
1879 THIN, GEORGE, M.D., 22, Queen Anne-street, W. (C. 1889-90.)
L1852 THOMPSON, Sir HENRY, 35, Wimpole-street, W. (S. 1859-63. C. 1865-7. V.-P. 1868-70.)
1891 THOMSON, HENRY ALEXIS, M.D., 2, Coates-crescent, Edinburgh.
1884 THOMSON, JOHN, M.B., C.M., 18, Walker-street, Edinburgh.
1892 THORBURN, WILLIAM, B.S., 96, Mosley-street, Manchester.
1874 THORNTON, JOHN KNOWSLEY, M.B., 22, Portman-street, W.
1872 THORNTON, WILLIAM PUGIN, 35, St. George's-road, Canterbury.
1880 TIRARD, NESTOR ISIDORE, M.D., 28, Weymouth-street, W.
1884 TIVY, WILLIAM JAMES, 8, Lansdowne-place, Clifton, Bristol.
1856 TOMES, Sir JOHN, F.R.S., Upwood Gorse, Caterham, Surrey. (C. 1867-9.)
1882 TOOTH, H. H., M.D., 34, Harley-street, W. (C. 1892-3.)
1886 TOTSUKA, KANKAI.
L1872 TOWNSEND, THOMAS SUTTON, 68, Queen's Gate, S.W.
1888 TREVELYAN, E. F., M.D., Seamen's Hospital, Greenwich, S.E.
1881 TREVES, FREDERICK, 6, Wimpole-street, W. (C. 1887-90.)
1851 TROTTER, JOHN W., Bossall Vicarage, York. (C. 1865-9.)
1859 TRUMAN, EDWIN THOMAS, 23, Old Burlington-street, W.
1888 TUBBY, ALFRED HERBERT, M.B., 39, Finsbury-circus, E.C.
1867 TUCKWELL, HENRY MATTHEWS, M.D., 64, High-street, Oxford.
1858 TUDOR, JOHN, Dorchester, Dorset.
L1875 TURNER, FRANCIS CHARLEWOOD, M.D. (HON. SECRETARY), 15, Finsbury-square, E.C. (C. 1884-6. S. 1891-3.)
1882 TURNER, GEORGE ROBERTSON, 49, Green-street, W.
1863 TURNER, JAMES SMITH, 12, George-street, Hanover-square, W.
1890 TURNER, WILLIAM ALDREN, M.B., 13, Queen Anne-street, W.
1858 TURTLE, FREDERICK, Clifton Lodge, Woodford, Essex.
1880 TYSON, WILLIAM JOSEPH, M.D., 10, Langhorne-gardens, Folkestone.

1867 VENNING, EDGCOMBE, 30, Cadogan-place, S.W.
1889 VOELCKER, ARTHUR FRANCIS, M.D., B.S., 13, Welbeck-street, W.

L1867 WAGSTAFFE, WILLIAM WARWICK, B.A., Purleigh, St. John's-hill, Sevenoaks. (C. 1874, 1878-80. S. 1875-7.)
1885 WAKLEY, THOMAS, jun., 5, Queen's-gate, S.W.
1881 WALLER, BRYAN CHARLES, M.D., Masongill House, Cowan-bridge, Kirkby-Lonsdale.
1890 WALLIS, FREDERICK CHARLES, M.B., M.S., 26, Welbeck-street, W.

Elected

1888 WALSHAM, HUGH, M.A., M.B., B.C., 32, New Cavendish-street, W.

1873 WALSHAM, WILLIAM JOHNSON, M.B., C.M., 27, Weymouth-street, W. (C. 1881-3.)

1859 WALTERS, JOHN, M.B., Reigate, Surrey.

1892 WARD, ALLAN OGIER, M.D.Edin., Lansdowne House, Tottenham.

1892 WARING, HOLBURT JACOB, M.B., B.S., 15, Upper Brook-street, W.

1889 WASHBOURN, JOHN WYCHENFORD, M.D., Guy's Hospital, S.E.

1877 WATERHOUSE, CHARLES, M.B., M.C., Carl Ludwigstrasse, Währing, Vienna.

1891 WATERHOUSE, HERBERT FURNIVALL, M.D., C.M., 81, Wimpole-street, W.

1880 DE WATTEVILLE, ARMAND, M.A., M.B., 30, Welbeck-street, W.

1892 WEAVER, FREDERICK POYNTON, M.D., Cedar Lawn, Hampstead Heath, N.W.

1890 WEBB, CHARLES FREER, M.D., New-street House, Basingstoke.

L1858 WEBER, HERMANN, M.D., 10, Grosvenor-street, W. (C. 1867-70. V.-P. 1878-80.)

1876 WEIR, ARCHIBALD, M.D., St. Mungho's, Great Malvern.

1864 WELCH, THOMAS DAVIES, M.D., Fairmount, Frith-hill, Godalming, Surrey.

1853 WELLS, Sir THOMAS SPENCER, Bart., 3, Upper Grosvenor-street, W. (C. 1865-8. V.-P. 1876-7.)

1892 WESBROOK, FRANK F., M.D.(Winnipeg), Cambridge.

L1851 WEST, CHARLES, M.D. (C. 1856-7.)

1877 WEST, SAMUEL, M.D., 15, Wimpole-street, W. (C.1884-6, 1891-3. S. 1889-90.)

1888 WETHERED, FRANK J., M.D., 34, Queen Anne-street, W.

1891 WHEATON, SAMUEL WALTON, M.D., 52, The Chase, Clapham Common, S.W.

1867 WHIPHAM, THOMAS TILLYER, M.B., 11, Grosvenor-street, W. (C. 1880-2.)

1869 WHIPPLE, JOHN H. C., M.D., Army Medical Staff.

1877 WHITE, CHARLES HAYDON, 20, Shakespeare-street, Nottingham.

1891 WHITE, GILBERT B. MOWER, M.B., B.S., 105, Gower-street, W.C.

1881 WHITE, WILLIAM HALE, M.D., 65, Harley-street, W. (C. 1888-90.)

1886 WHITE, WILLIAM HENRY, M.D., 43, Weymouth-street, W.

‡1868 WHITEHEAD, WALTER, 24, St. Ann's-square, Manchester.

1877 WHITMORE, WILLIAM TICKLE, 7, Arlington-street, S.W.

1870 WICKSTEED, FRANCIS WILLIAM, Chester House, Weston-super-Mare.

1869 WILKIN, JOHN F., M.D., M.C., New Beckenham, Kent.

1871 WILKINSON, J. SEBASTIAN, New Zealand.

1855 WILKS, SAMUEL, M.D., F.R.S. (TRUSTEE), 72, Grosvenor-street, W. (C. 1857-60. V.-P. 1869-72, 1883-5. P. 1881-2.)

1879 WILLCOCKS, FREDERICK, M.D., 14, Mandeville-place, W.

1886 WILLETT, EDGAR WILLIAM, M.B., 25, Welbeck-street, W.

1869 WILLIAMS, ALBERT, M.D. (Travelling).

Elected

‡1858 WILLIAMS, CHARLES, 48, Prince of Wales-road, Norwich.
1866 WILLIAMS, CHARLES THEODORE, M.D., 2, Upper Brook-street, W. (C. 1875-8.)
1881 WILLIAMS, DAWSON, M.D., B.S., 25, Old Burlington-street, W. (C. 1893.)
1872 WILLIAMS, JOHN, M.D., 63, Brook-street, W. (C. 1878-80.)
1864 WILLIAMS, W. RHYS, M.D., Linden House, Bertie-road, Leamington.
1881 WILLIAMS, W. ROGER, 28, Winckley-square, Preston.
1876 WILLIAMSON, JAMES MANN, M.D., Ventnor, Isle of Wight.
1863 WILLIS, FRANCIS, M.D., The Spa, Braceborough, Stamford.
1889 WILSON, ALBERT, Leytonstone, Essex.
1888 WILSON, CLAUDE, M.D., C.M., 6, York-road, Tunbridge Wells.
1859 WILSON, EDWARD THOMAS, M.B., Montpelier-terrace, Cheltenham.
1859 WILSON, ROBERT JAMES, 7, Warrior-square, St. Leonards-on-Sea.
1891 WILSON, THEODORE STACEY, M.B., C.M., 65, Temple-row, Birmingham.
‡1861 WINDSOR, THOMAS, Medical Library, Boston, Mass., U.S. [care of B. F. Stevens, 4, Trafalgar-square, W.].
1889 WINGRAVE, V. HAROLD WYATT, 7, Taviton-street, Gordon-square, W.C.
1874 WISEMAN, JOHN GREAVES, Dearden-street, Ossett, Yorkshire.
1865 WITHERBY, WILLIAM H., M.D., Pitt-place, Coombe, Croydon.
1887 WOOD, T. OUTTERSON, M.D., 40, Margaret-street, W.
1876 WOOD, WILLIAM EDWARD RAMSDEN, M.A., M.D., The Priory, Roehampton, S.W.
1883 WOODCOCK, JOHN ROSTRON, 155, Hagley-road, Birmingham.
1883 WOODHEAD, GERMAN SIMS, M.D., Beverley, Nightingale-lane, Balham, S.W. (C. 1891-3.)
1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.
1884 WORTS, EDWIN, 6, Trinity Street, Colchester.
1869 WYMAN, WILLIAM S., M.D., Red Brae, 18, Putney-hill, S.W.
1890 WYNNE, EDWARD T., M.B., County Asylum, Rainhill, Prescot,
1884 WYNTER, WALTER ESSEX, 30, Upper Berkeley-street, W.

1872 YOUNG, HENRY, M.B., Monte Video, South America.

ANNUAL REPORT OF COUNCIL,

1892 93.

PRESENTED AT THE ANNUAL MEETING, MAY 16TH, 1893.

YOUR Council has to report the admission of nineteen new members, of whom fourteen are resident and five non-resident. There have been six resignations and six deaths. The list of deaths comprises Dr. James Anderson, Dr. Conway Evans, Dr. Hope, Dr. Bernard Lawson (New Zealand), Dr. James Ross (Manchester), Dr. Sibley, and Dr. John Davies Thomas (Adelaide). Thirteen members have compounded (£101 17*s.*).

Two General Meetings have been summoned during the Session, and have sanctioned certain changes in the Society's Laws proposed by the Council. These changes relate, firstly, to the composition fees, which experience had shown to be considerably lower than compatible with the financial prosperity of the Society; and secondly, to the method of electing the Society's officers. They will henceforth stand in the Laws of the Society.

The financial report for the year must be regarded as highly satisfactory. The income shows an increase of £85 2*s.* 2*d.*, the expenditure a diminution of £116 4*s.* 5*d.* A balance was left of £247 17*s.* 2*d.* This has enabled the Council to purchase £170 Consols, which will rather more than replace the sum which had to be sold last year.

The apparent increase in income was, however, largely due to composition fees received in lieu of annual subscriptions, and must therefore be regarded as capital.

Signed on behalf of the Council,

G. M. HUMPHRY,
President.

May 2nd, 1893.

THE PATHOLOGICAL SOCIETY OF LONDON,

Cr. *In Account with the Treasurer*, WILLIAM CAYLEY, ESQ., M.D., *45th Session*, 1892–93. Dr.

From 1st May, 1892, *to 30th April*, 1893.

	£	s.	d.	£	s.	d.
By *Balance* in hand, May 1st, 1892				120	19	7
By *Subscriptions:*						
395 paid direct ..... }	432	12	0			
17 „ through Bank ..... }						
412						
12 Admissions at 21*s.*	12	12	0			
2 Non-Resident Compositions at £2 2*s.* ..	4	4	0			
2 „ „ at £3 3*s.* ..	6	6	0			
13 Life Composition Fees (various).....	101	17	0			
				557	11	0
By *Sale of Transactions:*						
Per Publisher	47	5	7			
„ Assist. Sec.	12	1	6			
				59	7	1
By *Dividends*				26	16	4
				£764	14	0

	£	s.	d.	£	s.	d.
To *Meetings:*						
Rent to Christmas, 1892	105	0	0			
Refreshments to May, 1892	36	15	0			
R. Coldrey do. do.	7	10	0			
Microscopes to April, 1893	12	16	6			
				162	1	6
Transactions:						
Printing, Binding, &c., of Vol. XLIII.....	190	11	3			
Illustrations	79	13	0			
Index	3	3	0			
				273	7	3
Secretariat and Treasury:						
Assistant Secretary to May, 1892	31	10	0			
Commission to Collector do. do.	15	15	0			
Bank Charges April, 1893	1	14	7			
Petty Cash do. do.	1	5	6			
				50	5	1
Extraordinary Payments:						
Expenses of Meetings to Christmas, 1892 ..	12	5	0			
Assistant Secretary do. do. ..	15	15	0			
Index to Vol. XL	3	3	0			
				31	3	0
Purchase of "Consols" (£170)				168	11	6
				685	8	4
Balance in hand				79	5	8
				£764	14	0

Audited and found correct, May 10th, 1893,

W. HALE WHITE, M.D.,
J. WARRINGTON HAWARD, } *Auditors.*

W. CAYLEY, M.D., *Treasurer*,
May 10th, 1893.

F. CHARLEWOOD TURNER, M.D.,
S. G. SHATTOCK, } *Hon. Secs.*

Amount of Investments in hands of Trustees—Consols £1170.

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REPORT.

SESSION 1892-1893.

I. DISEASES, ETC., OF THE ORGANS OF RESPIRATION.

1. *A case of chylous pleurisy and ascites.*

By H. G. Turney, M.B., introduced by S. G. Shattock.

For the use of the clinical notes of this case I am indebted to Dr. Ord.

The patient, Arthur S—, aged 54, was admitted into St. Thomas's Hospital on March 11th, 1893. For six months before this he had been vomiting his food; this latterly had been growing worse, and had caused him to apply for admission. During the past five weeks he had noticed the successive appearance of lumps, first in the right side of the neck, then in the left, and finally in the axillæ and groins. There was no history of syphilis, and no previous illness of any importance.

On admission he was pale and emaciated. The chest was barrel-shaped, and moved badly; rhonchi were audible over both lungs. There were no signs of ascites, and the liver and spleen were not enlarged; the liver, however, appeared to be displaced slightly downwards, its edge descending 1½ inches below the costal arch. The legs were moderately œdematous. The urine was of sp. gr. 1020, and contained neither albumen nor sugar. The pupils were equal and active, and the voice was unaffected. The cervical and submaxillary glands on both sides were considerably enlarged, more so on the right side than the left. There was also swelling, but less marked, of the supra-clavicular, axillary,

and inguinal groups of glands. Tenderness was completely absent, and the glands remained quite individualised.

The patient lived till April 2nd, and between the date of his admission and his death few fresh symptoms developed. The vomiting continued; the glands increased in size; signs of fluid appeared in the left side of the chest; and some abductor paralysis of the larynx was observed. His weight remained almost stationary, but his weakness steadily increased. The urine remained free from albumen, and the temperature was always subnormal.

The autopsy was performed thirty hours after death. The body was that of an emaciated man (weight 6 st. 5 lbs.), with œdema of back and legs.

The abdomen contained about 4 ounces of milky fluid. Under the microscope this exudate appeared as a collection of fat globules of various size, and the "molecular basis." The peritoneum was whiter than usual, and faintly marked with white lines. There were no signs of inflammation. Stomach: the pyloric orifice was considerably narrowed by new growth, its walls being increased in thickness to quite two thirds of an inch. The thickening stopped short at the duodenum, but extended for an inch, or an inch and a half, on to the surface of the stomach, where it gradually faded away. In the pyloric part the surface was slightly ulcerated. The new growth was firm on section, and of a semi-translucent yellowish appearance. Microscopical examination proved it to be composed of large epithelial cells of a spheroidal shape, mostly filled with colloid material. These cells had a glandular arrangement, were continuous with the glandular elements of the mucosa, and infiltrated the muscular coat. The inner surface of the stomach was thickly strewn with nodules of a similar structure, varying in size from a hazel-nut downwards. The stomach was not dilated, and had not formed adhesions to other viscera. The cardiac orifice was unaffected and the œsophagus healthy.

The contents of the intestine were of the ordinary description. The villi were greatly enlarged, their appearance being suggestive of a two or three days' beard. The lymphatics, especially in the ileum, were much distended, and their course could be seen marked out in white lines under the surface of the peritoneum.

The mesenteric glands were large and soft, but not infiltrated with growth.

The liver and spleen were normal in appearance.

The kidneys were small (weight 7 oz.), but healthy.

The bladder contained clear urine.

The right pleura contained two pints of fluid having the appearance of milk. Microscopically also it was identical with that found in the peritoneum. The pleural membrane was white and opaque. The antero-inferior angle of the lower lobe of the right lung was firmly adherent; the included lung tissue was cirrhotic, and the bronchial tubes in it dilated. The lower lobe was collapsed. At the apex were two calcareous nodules with some scarring of the superjacent pleura. The left pleura contained one pint of turbid serum, with some fat held in suspension, but without the milky aspect of the fluid found on the other side.

Apart from some collapse at the base, the left lung was healthy.

The pericardium contained three ounces of clear yellow serum, Its outer surface was covered with ramifying white lines representing dilated lymphatics.

The heart was normal.

Lymphatic system.—The lymphatics in the right pectoral muscles were greatly dilated, their contour showing alternate contractions and dilatations corresponding to the valves. Nearly all the superficial glands in the body were enlarged; amongst them were those on both sides of the neck, in both axillæ, and the groins. A number were taken at random and examined microscopically. These all proved to be infiltrated with new growth of the same nature as that affecting the pylorus.

The thoracic duct was dilated in its whole course, and surrounded by an unusual amount of œdematous connective tissue. It was patent to its termination by two orifices in the junction of the left subclavian and internal jugular veins; being cut across just above this, some milky fluid spurted out. The orifices of the duct in the vein were surrounded and blocked up by dry, partially decolourised, adherent clot, which almost immediately on either side tailed off into the ordinary soft black coagulum. The pathological clot, though adherent, could be separated fairly easily from the wall of the vein, leaving the intima unaffected. Neither the subclavian nor internal jugular veins, though surrounded by large glands, were compressed by or adherent to them.

The obstruction of the thoracic duct was clearly due in this case to the thrombosis at its outlet. What, then, was the cause of this thrombosis? It was so clearly localised about the orifice of the

duct that one is impelled to ascribe it to the lodgment there of a foreign body. The preceding account has shown that the new growth was disseminated throughout the lymphatic system, and it seems fair to suppose that a fragment carried along the duct may have been arrested at this spot. The bifurcation of the duct immediately above its termination may possibly have produced a relative stricture. In the absence of any disease of the veins or pathological stricture of the duct, it is difficult to see what other explanation can be suggested.

If a cancerous embolus did cause the mischief, its part must have been a passive one, as there was no indication of any new growth in the vein; the intima was unaltered, and the clot appeared of the same composition throughout. Unfortunately no microscopical examination of the clot was made; it was originally very small, and broke up in the process of inspection.

The nearest parallel to this case is one recorded by Dr. Cayley in the seventeenth volume of this Society's 'Transactions.' His description is as follows:—"At the junction of the thoracic duct with the subclavian vein the former suddenly became much narrowed, and its coats thickened, and just at its mouth a fibrinous granular vegetation was attached to the lining membrane of the vein, which almost completely obstructed the opening of the duct."

Another case was reported by Dr. Sidney Martin in the forty-second volume of the same 'Transactions;' in this the cause of the thrombosis was primary contraction of the veins.

In nearly every case of obstruction of the duct hitherto recorded with reference to the question, rupture has occurred, generally at the receptaculum chyli. Ligature of the duct in animals is said to be invariably and speedily followed by rupture.

That the duct in the case now described was not ruptured seems certain, for not only did it contain fluid just above its termination, but fluid under considerable pressure. From the small amount of fluid in the peritoneal cavity the receptaculum chyli may undoubtedly be regarded as having been intact. Moreover, the duct having been dissected out to its termination, the seat of rupture, had one been present, could hardly have been missed. Both the clinical history and the pathological condition point to a rapid, rather than a gradual onset of the obstruction, rendering the fact of non-rupture the more noteworthy. *May 16th*, 1893.

2. *Primary cancer of pleura.*

By R. G. Hebb, M.D.

The patient, a female aged 46, was admitted into the Westminster Hospital under Dr. Donkin on December 13th, 1892, for "weakness." During the past two months she had been getting thin, and also suffered from a dry cough, dyspnœa, and debility. Family history and personal habits good. She had never had any previous serious illness, but had not been feeling well for the past two or three years. There was complete dulness with absence of movement and respiratory sounds over right thorax; there was no contraction of thorax. Physical signs remained exactly same after tapping, when 29 oz. of brownish-red fluid were removed, in which one large epithelioid cell showing a "cancer body" was observed. She gradually sank, and died January 30th, 1893.

A *post-mortem* examination was made twenty-three hours after death. Body emaciated. No difference between the two halves of thorax. Heart and left lung not displaced. Right pleura is greatly thickened, and in the sac are from two to three pints of brownish-red turbid fluid, and much brownish-yellow thin soft clot. The internal surface is ragged and rough from deposit of clot, and dark red from blood-staining and hyperæmia. The lung, which is spheroidal in shape, lies in the upper half of the costo-vertebral groove. It is about the size of a large orange; it is soft, fleshy, and much pigmented. It contains an irregular white firm deposit about the size of an almond, which lies close to, and in connection with, the visceral pleura in front. The parietal pleura is everywhere greatly thickened, especially at apex, along costo-vertebral groove, and over the diaphragm. In some places it is an inch thick. On section it is quite white, very tough, and resembles dense fibrous tissue. The right half of the diaphragm is infiltrated by the growth, which appears on the peritoneal surface as uneven prominences, either continuous with the pleuro-diaphragmatic infiltration or as flat isolated separable deposits. Over an area as large as a crown piece, the liver capsule on the upper surface of the right lobe exhibits a similar infiltration, or thickening. There are numerous secondary deposits over the peritoneum

and left pleura; these are round, hard, pearly little nodules, largest on omentum, bladder, and mesentery, but none are bigger than a lentil or small pea. Of the lymphatic glands a few of the mediastinal are infiltrated, the deposit being firm and hard. All other parts and viscera were not noteworthy.

The heart, liver, spleen, kidneys, uterus, ovaries, mammæ, bones, gastro-intestinal tract, fauces, larynx, and thyroid showed nothing abnormal.

Microscopical examination of the pleura shows that it is composed of fibrous tissue tunnelled up by channels lined by epithelioid cells. In places the cells are in single column, in others the alveoli are lined by a double layer of cuboidal epithelium. The internal surface is covered with organising blood-clot.

The grounds on which I consider this case to be one of primary cancer of the pleura are, that the pleura is the part principally—indeed, almost alone affected, and that the secondary growths are extremely small, and are limited to the peritoneum, left pleura, and a few mediastinal lymphatic glands. It is necessary to allude to this because primary cancer of the pleura is a rare disease, and the statement usually gives rise to objections and scepticism; but in this particular instance no other part could claim to have been the original starting-point.

The only other case I have seen was that exhibited by Dr. G. N. Pitt in 1888, and recorded in volume thirty-nine of the Society's 'Transactions.' Very similar cases will be found recorded by Coats in the 'Glasgow Medical Journal' of July, 1889, and in the 'Lancet' of November, 1885 (p. 945), by Dr. W. Collier. See also Dieulafoy, 'Soc. méd. Hôp. de Paris,' 1886, p. 36; and Gougenheinn, 'Soc. méd. Hôp. de Paris,' 1886, p. 161.

N.B.—An excellent idea of the cancer body found in the fluid drawn off from thorax may be seen in pl. lxxiv, Beale's 'Microscope in Medicine,' 4th edition. *April 18th*, 1893.

3. *Stenosis of left bronchus caused by dilation of left auricle.* (*Card specimen.*)

By W. Lee Dickinson, M.B.

From a woman, aged 20, who died in February, 1893, in St. George's Hospital under the care of Dr. Cavafy.

The heart was greatly enlarged, the two layers of the pericardium adherent, the edges of the mitral valve thickened, and the valve funnel-shaped, though not much stenosed.

The left auricle was greatly dilated, and by pressing upwards had considerably narrowed the lumen of the left bronchus. The lower lobe of the left lung was partly hepatised.

A specimen similar to this is described by Dr. H. H. Taylor in the fortieth volume of the 'Transactions.' *March 21st*, 1893.

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4. *Emphysematous lesions of the lungs.*

By F. Charlewood Turner, M.D.

My first specimen is the right lung of a girl, aged 11, who had been run over in the street, and died shortly after her admission to the London Hospital from hæmorrhage into the peritoneal cavity, due to an extensive laceration of the liver, and shock.

Along the inferior border of the lower lobe, at its anterior part, is a fringe of subpleural emphysematous blebs of unequal size, which project from both the under and outer surfaces of the lung, more numerously below. The largest, about the centre of the row, was about the size of a haricot bean. There were a few ecchymoses on the surface of the lungs.

The liver was extensively lacerated in an antero-posterior direction between the lobes, especially on the under surface, showing that it had been crushed between the spinal column and the wheel passing over the epigastric region. There was much blood in the

peritoneal cavity, and some also extravasated behind the membrane, and in the posterior mediastinum. There was no external bruising of the body.

The second specimen shows the lungs of a female infant, one year old, who died with tubercular pyelo-nephritis affecting the right kidney and extending down the ureter to the bladder, which was affected on that side especially, and with miliary tuberculosis of the lungs and pleura with caseous bronchial glands, and bronchitis.

The lungs exhibit a high degree of injury to the anterior parts of the upper lobes from strenuous respiratory efforts or straining with cough. Conspicuous tracts of interstitial emphysema run forwards and downwards over the surface of the middle and upper lobes of the right lung towards their anterior borders, and on the internal surface of the lung is a large tract of interstitial emphysema along the line of junction of these lobes.

There is also a similar subpleural laceration of the pulmonary tissue of the upper lobe just in front of the root of the lung, on the same surface. In the left lung there is a tract of interstitial emphysema extending backwards and upwards from a notch in the anterior border of the upper lobe, in a position corresponding with that between the middle and upper lobes of the right lung.

All these appear to be recent effects of injury to the lung from strenuous respiratory exertion or straining in coughing, but near the tip of the middle lobe and at its inferior border is a large loculated emphysematous cavity with thick walls, and of older date.

There are also several conspicuously dilated vesicles, isolated and clustered, over the surface of the upper lobes of the lungs, and in the pleura over some of them opaque spots like miliary tubercles are seen.

There are some caseous glands at the roots of the lungs, and at the root of the left lung in the upper part of the lower lobe is a cavity with a caseous sequestrum loose within it, apparently from suppuration of a caseous gland.

The infant from whom these organs were obtained was admitted into the London Hospital for marasmus, with a history of diarrhœa since being weaned at nine months. The bowels were irregular in hospital, and the child was very fretful and cried much; but had no cough or dyspnœa, according to the notes made.

Remarks.—That full inspiratory expansion of the thorax causes a disproportionately great expansion of the thin borders of the

lungs is a necessary result of the configuration of the pleural cavities, and of the greater expansibility of the lungs as compared with the mediastinal organs.

That compression of the fully expanded lungs by forcible action of the muscles of expiration with closed glottis, as in straining or coughing, causes emphysematous lesions of the anterior borders of the lungs of more severe degree than is producible by inspiratory effort, is seen (1) from the fact that under such circumstances a further projection of the sternum than is producible by full inspiration occurs, and (2) from the consideration that the expiratory compression of the contents of the chest must at the same time cause a partial emptying of the heart cavities, which the preceding inspiratory effort must, on the other hand, have caused to become over-filled. Expiratory efforts with closed glottis after full inspiratory expansion of the chest must then, on both accounts, cause a further separation of the anterior chest wall from the heart, to be filled up by an increased distension of the edges of the lungs, thrust forward between them.

The facts prove that expiratory compression of the expanded lungs causes the severest injury to the anterior border of the lungs, but they do not prove that this result is due wholly, or even mainly, to insufficiency of the support afforded to the sternum to sustain the compressing force of the powerful expiratory muscles upon the lungs and other contents of the chest. The support afforded to the sternum by its connection with the spine by the ribs, and by the powerful bracing up of the rectus abdominis and other expiratory muscles, would, indeed, appear to be considerable.

It appears to me that the *mechanical advantage* afforded by the wedge-like shape of the anterior borders of the lungs to the distending force in this direction of the compressed air in the lungs, subjected to the action of these powerful muscles, is a potent factor in causing protrusion of the sternum and costal cartilages and compression and thrusting backwards of the heart and pericardium, and the emphysema of those parts so produced.

The main cause of the great emphysematous expansion of the anterior borders of the lungs under such circumstances is, of course, the fixation of the lung at its root, so that it cannot move bodily forwards in adaptation to the altered dimensions of the cavity, into the expanded angles of which the compressed and

imprisoned air can only get by distending or bursting the borders of the lungs.

The subpleural laceration of the lung tissue on the inner surface of the right lung, immediately in front of the root of the lung in my second specimen, to which attention was directed, is, I think, interesting as evidence of the strain upon the lung tissue at this part when the anterior borders are driven forward by the air compressed in the closed cavity.

That the severe lesions of the anterior parts of the lungs seen in my second specimen are attributable to the effect of powerful expiratory efforts acting on diseased pulmonary tissue, after full inspiration, in straining efforts, or in coughing, is, I think, certain, although the child would appear not to have been greatly troubled with cough.

The emphysematous insufflation of the lower border of the right lung shown in my first specimen, I at first supposed to have been also produced by protrusion, in the direction of least resistance, and of greatest mechanical advantage, of the air in the lung under high pressure from compression between the ground and the cart-wheel. But it seems to me more probable that, in this instance, the lesion was, in reality, due to dragging open of the angle between the diaphragm and the chest wall in the lateral region, by the combined effect of a lateral bulging of the chest, flattened in an antero-posterior direction, and of a simultaneous thrusting down of the liver and diaphragm by the crushing effect of a wheel passing over the epigastric region.

The specimen shows the amount of injury which may be sustained by a healthy lung under such conditions. But although expiratory compression of the fully expanded lungs is undoubtedly the direct cause of emphysematous lesions of the anterior borders of these organs of the most severe degree, the lesions inflicted upon the lungs by this agency can never have a very important effect in impairing the functional capacity of the organs.

The injuries to the anterior border of the lungs, so conspicuous in my second specimen, are of inconsiderable extent in comparison with the whole volume of the pulmonary tissue, and are probably, for the most part, recoverable—though not wholly so, as appears from the specimen. And the same may be said of any injury to the surface of the lungs similarly resulting from bulging of the supra-clavicular or intercostal spaces.

Supposing the bulging of the intercostal spaces to be equivalent to the change of the transverse section of the pleura from a straight line to a semicircle, and supposing further that the lung were firmly adherent to the chest wall, so that the compressed lung could not slip into the bulging spaces, the air-vesicles over the bulging area would then be, for the moment, distended on an average to one and a half times their natural size in that direction: in a direction parallel with the ribs the over-distension of them would be inconsiderable, if anything at all. If the compressed lung were non-adherent and could bulge freely between the ribs, the distension would be distributed over the whole extent of the vertical section of the lung, and would correspond in extent with the increase of that only.

At the apex a degree of bulging from a flat membrane to a hemisphere would be attended at most by a distension of the surface vesicles to one and a half times their normal size.

Such considerations show, I think, that expiratory compression of the lungs in straining or coughing can have little to do, directly at least, with the occurrence of that general emphysematous condition of the lungs with immobile barrel-chest, on which the discomfort, disabilities, and dangers, clinically characteristic of that condition, are attendant.

This general over-expansion of the lungs is attributable directly to inspiratory effort, aggravated by expiratory distension of the pulmonary tissue at certain parts only.

But the real cause of the permanent emphysematous expansion of the lungs in the barrel-chest must be some defective nutritive state of the pulmonary tissue itself, or of the finer air-passages—probably of both. It is difficult to suppose that any inspiratory expansion of the healthy lungs could have any seriously injurious effect upon them; for if the whole lung is freely expansible, the utmost possible inspiratory over-expansion of any individual vesicles, except at the borders, must be very slight in degree, being measured by the proportionate increase of the dimensions of the pleural cavity in different directions; no breaking down of the interalveolar septula could be so brought about in them.

"Emphysema," as a pathological lesion of the whole of the lung tissue, must, then, be dependent upon conditions in the lungs causing the expansive force of inspiration to bear especially upon certain parts of the vesicular tissue, as in the case of a bronchitis

affecting the smaller tubes, and preventing or greatly impeding the entrance of air into many lobules throughout the lungs; or, on the other hand, causing a greatly weakened resistance of certain of the air-vesicles, or of certain clusters of air-vesicles, to the expansive force of inspiration, leading to their disproportionate expansion, as perhaps in the case of the conspicuous vesicles often seen over the surface of lungs affected with miliary tuberculosis, with grey miliary tubercles appearing as opaque spots in their centre or immediately contiguous to them, some of which are to be seen in my specimen.

In the case of those emphysematous patients who have not suffered from bronchitis to their knowledge, it seems most probable that the lung lesion was due to some repeated catarrh of the smaller bronchial tubes attended with little annoyance, or possibly to some nutritive defect, dependent, it might be, on renal cirrhosis, so far interfering with the elasticity and resilience of the pulmonary tissue as to lead to the occurrence of the lesion.

November 1*st*, 1892.

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5. *Gangrenous pneumonia fatal by intra-pleural hæmorrhage.* (*Card specimen.*)

By E. C. Perry, M.D.

A right lung, a portion of the upper lobe of which has been removed to exhibit, towards its base, a ragged cavity about an inch in diameter. The pulmonary tissue around the cavity is consolidated by pneumonia. Within the cavity the blue rod indicates a large branch of the pulmonary artery, the ulceration of which led to fatal hæmorrhage. A communication between the excavation and the pleural cavity is marked by a red rod. There is recent lymph upon the pleura.

William L—, aged 45, was admitted into Guy's Hospital under Dr. Perry for hæmoptysis and pyrexia with dulness at the lower part of the front of the right chest. He died the day after his admission with symptoms of internal hæmorrhage. At the autopsy

52 oz. of clotted blood were found in the right pleural cavity. It seems probable that the pneumonia was consequent upon an attack of epidemic influenza.

No similar case is recorded in the 'Transactions' of the Society.

October 18*th*, 1892.

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6. *Lymphadenoma of the lungs and other organs.*

By Arnold Chaplin, M.D.

The specimens were removed from a woman, aged 32, who had a history of good health until two years previously, when she had an attack of influenza. She applied for treatment complaining of the ordinary symptoms of anæmia, the dyspnœa being excessive on the slightest exertion. There were large masses of glands in the neck on each side, in the axillæ, and groins. They were not adherent to the skin, and freely moveable upon one another. At the right apex there was deficient movement, and the percussion note was absolutely dull down to the fourth rib, and extended to some distance over the sternum. Over this area there was loud bronchial breathing and pectoriloquy. Subsequently the liver and spleen became enlarged. The temperature during observation varied between 99° and 102°.

At the autopsy the whole of the superior and middle lobes of the right lung were infiltrated with a hard growth, which had originated around the bronchi, but presented no caseating centres. This growth had connection with an enlarged mass of glands in the anterior mediastinum. There was hardly any lung tissue left in the parts infiltrated with the growth. The bronchial glands, the lumbar glands, and the superficial glands of the neck, axillæ, and groins were all enlarged, but not caseous. Some nodules of caseating lymphatic tissue were present in the left lung.

The spleen weighed 14½ oz., and presented a typical lymphadenomatous aspect.

The diagnosis of this case had to be made between sarcoma and lymphadenoma. For some time this was doubtful, but the general enlargement of the glands all over the body, the enlargement of the

spleen, and the continued elevation of temperature enabled one easily to decide in favour of lymphadenoma.

The microscopical appearances did not show that the growth was a sarcoma. *May 16th*, 1893.

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7. *Growth of the mediastinum implicating the heart and opening into both the œsophagus and the aorta.*

By W. HALE WHITE, M.D.

MICHAEL H—, aged 37, was admitted into Guy's Hospital on January 20th, 1893. Seven months ago he had slight hæmoptysis, but this did not trouble him, and he felt well and able to work till three months ago, when he began to suffer from weakness and shortness of breath. He has gradually since then lost his appetite and grown thinner, and now he feels very ill. During this time he has occasionally had some pain in the left chest, and some difficulty of swallowing, but these have now entirely disappeared.

On admission.—Is thin and wasted. The left chest looks more prominent than the right, but it does not measure more. There are physical signs indicative of fluid filling the whole of the left chest, and the insertion of an exploring needle withdrew some fluid, clear but slightly blood-stained. The veins about the upper part of the left chest are slightly more marked than those on the right. There is a cough which is somewhat brassy. The apex of the heart cannot be defined. Over the aortic area a to-and-fro murmur can be heard; the systolic murmur is conducted upwards to the right, and the diastolic down the sternum. The radial pulses are equal; no enlarged glands can be felt anywhere. The diagnosis was aortic disease, together with malignant growth in the left posterior mediastinum.

January 24th.—A pericardial rub was heard to-day. This gradually became louder day by day for a few days, and then slowly disappeared. Coincident with the appearance of the peri-

cardial rub, the cardiac dulness increased to the right of the sternum.

February 18th.—Till to-day the patient has persistently refused to have his chest aspirated, but to-day a few ounces of fluid were withdrawn. He complains of difficulty of swallowing, and his dyspnœa is greater, and the left pupil is slightly smaller than the right.

20th.—At midnight the patient suddenly brought up a large quantity of blood, and in few minutes he was dead.

Autopsy.—A small mass of growth was found behind the origin of the left sterno-mastoid. On opening the thorax a mass of hard malignant growth was seen in the upper and left parts, invading or displacing the surrounding structures.

The upper lobe of the left lung was displaced outwards and largely invaded, and collapsed from obstruction to the bronchus; a portion of it was gangrenous and very foul-smelling. The base was compressed by a localised collection of blood-stained fluid (24 oz.). The pleura was much thickened everywhere, and the parietal layer was very loosely attached, coming away with the lung.

The pericardial sac was extensively involved, and the growth had caused plastic pericarditis obliterating the cavity. The adhesions were recent, and were easily broken down with the finger.

The left auricular wall was so largely implicated as to be formed almost entirely of new growth, and into its cavity some nodules projected. They varied in size from a pea to a hazel-nut, and were cystic. The right side of the heart was healthy; the wall of the left ventricle was fatty. The pulmonary artery and veins were surrounded and slightly constricted by growth.

The aorta was markedly constricted, thus accounting for the murmur. The aortic valves were perfectly healthy. Half an inch below the origin of the left common carotid the aorta was ulcerated through, the opening being about as big as a fourpenny piece, and communicating with a gangrenous cavity in the growth.

The œsophagus was extensively involved and ulcerated. At one place the mucous membrane was ulcerated right through, the perforation communicating with the same gangrenous cavity as that with which the aorta was continuous, thus explaining the sudden death from hæmorrhage.

The left bronchus was surrounded, pressed upon, and completely occluded; the left recurrent laryngeal nerve was involved.

The lower part of the œsophagus, the stomach, the duodenum, and other parts of the intestine were filled with blood-clots, some of which formed very complete casts of the intestine and its valvulæ conniventes. The rest of the body was healthy. The growth was a round-celled sarcoma.

The following points seem to me to render this case worthy of being placed on record:—1. The perforation of the aorta causing death by hæmorrhage. This is a very rare event, for, as is well known, arteries are hardly ever attacked by malignant growth. In 1885 I made a *post-mortem* examination on a case of epithelioma of the œsophagus, which had ulcerated into the aorta exactly opposite the left subclavian artery. The aperture was the size of a pea. The stomach contained a quantity of blood-clot, which had no doubt passed from the aorta into the stomach through the aperture above mentioned. This case has been recorded by Dr. Frederick Taylor.[1] He quotes from Wilks and Moxon to show that when malignant growth ulcerates into an artery, it is usually sloughing; and no doubt, as the growth recorded in the present communication was gangrenous, it was by process of sloughing that it opened into the aorta. I need not go into the literature of the subject, as Dr. Frederick Taylor has done that; but to show the rarity of perforation of the aorta from growth, I may mention that Allman, quoted by Dr. Taylor, only met with it four times in nearly 7,000 *post-mortem* inspections. Among the cases he collected, Dr. Taylor found that all were males aged between forty-two and sixty-five; profuse hæmorrhage was a common cause of death, and blood was very often found in the œsophagus, stomach, and intestines in considerable quantity. The perforation of the aorta usually took place in the lower half of the arch or the upper half of the thoracic aorta. It will be seen that my case, excepting that my patient was rather young, agreed with the facts brought out by Dr. Taylor's analysis.

2. The compression of the aorta so as to produce a murmur. This is a very rare event. It did not occur in any of the cases recorded by Dr. Vincent Harris,[2] nor does Hare[3] mention it in any of the 232 cases of malignant disease of the mediastinum which he

[1] "Some Cases of Malignant Disease of the Chest and Abdomen," 'Guy's Hospital Reports,' vol. xlix.

[2] "Intra-thoracic Growths," 'St. Barth. Hosp. Rep.,' vol. xxviii.

[3] 'Mediastinal Disease,' Philadelphia, 1889.

has collected. We, during the patient's life, considered, I think pardonably, that the murmur was due to aortic obstructive disease.

3. The invasion of the walls of the heart. This was in the present case very extreme. It is of unusual occurrence. Among Hare's 232 cases, it is mentioned in nine that the heart was implicated, but in some of these it is clear that the growths in it were secondary deposits, which would have been almost equally likely to be found had the primary seat of the disease been elsewhere than in the chest. We may, I think, conclude that in not more than 1 or 2 per cent. of the cases of malignant disease of the mediastinum does the growth spread into the walls of the heart. On the other hand, as is well known, pericarditis due to malignant disease of the mediastinum is fairly common.

May 2nd, 1893.

II. DISEASES, ETC., OF THE ORGANS OF CIRCULATION.

1. *Rupture of the heart in the insane.*

By Cecil F. Beadles.

The two ruptured hearts are from patients who died insane. The first specimen is from a woman, aged 65, who died suddenly in Colney Hatch Asylum, having been an inmate for thirty-three years. She had been insane at least forty years. On admission she was incoherent, and at times violent and dangerous to others; but since 1872 she had been demented and quiet. Health remained fair up to 1889, since which she had been more feeble, but not sufficiently so to keep her bed. On the morning of January 22nd, 1892, she was sitting at table taking her food, when she was seen to put her hand up to her head, and almost instantly fell back dead.

Post-mortem.—The heart, which is shown, was found surrounded by a black clot, on removing which it presented a rupture of the anterior wall of the left ventricle. The heart was exceedingly fatty. It was filled with black fluid blood, and weighed 8¼ oz. when empty. The muscular tissue of the heart is greatly thinned, and covered by a thick layer of fat externally. There is very little myocardium of the right ventricle remaining; and in the left ventricle, where the rupture has occurred, there is a general thinning of this coat. The muscular tissue is ploughed up internally in this vicinity, and the actual rent passes completely through in two places, ¾ inch apart. These are small and irregular, and about 1½ inches from the apex on the anterior surface. The flaps of the mitral and aortic valves are slightly thickened, and the aorta above the cusps is somewhat atheromatous.

The condition of the other organs was as follows:—Kidneys slightly enlarged, of dark colour, and granular on the surface. Liver and spleen healthy. Right ovary enlarged and calcified. Right pleuritic adhesions. Cerebrum and membranes fairly healthy, the only thing noticed being an apparent slightly smaller size of the left hemisphere. The left lobe of the cerebellum, however, was very small and exceedingly hard; there was only a trace of grey matter covering the convolutions, and in some parts there appeared to be none whatever. The right lobe was of natural size, but this and the central portion of the cerebellum was softer than natural. The grey matter here appeared normal. Weight of the whole cerebellum was 3½ oz., that of the entire brain being 44½ oz.

The second specimen is from a patient who was under the care of my colleague, Mr. E. R. Rouse. Mr. Rouse reported the case in the 'Lancet,' February 6th, 1892, but as his description was very brief, and as the specimen is of particular interest, perhaps I may be allowed to show and describe it more fully. It is from a man aged 54, who apparently lived for 146 hours after the actual rupture, or perhaps I should say partial rupture, took place. The heart is enlarged and very fatty; the deposit of fat is principally over the posterior surface, and at the auriculo-ventricular furrows. The ventricular walls are very thin, and the muscle substance pale and friable. The laceration is on the anterior surface of the left ventricle, is ⅝ of an inch in length externally, but somewhat less within, and is situated 1½ inches from the apex, the wall at this place being about ¼ inch thick. All the valves are fairly healthy, but the aorta is slightly atheromatous. At the autopsy the pericardial cavity contained about 16 oz. of clotted blood with serous fluid. The kidneys were in a state of chronic Bright's disease, the brain and membranes showed thickening and hardening with excess of fluid.

The patient was the subject of chronic mania with religious and other delusions, existing for ten years, but for the last eight years he had been quiet, and usefully employed. No mention is made of cardiac disease, and within a year of his death his health is said to have been good. He died on November 13th, 1891, when the following note was made:—"This evening, while sitting in the infirmary, he suddenly fainted away in his chair, and died almost immediately. He had been in his usual health till a week ago, working regularly in the carpenters' shop. On the 7th inst. he was amusing himself with other patients by kicking a football (not playing a match), and

after a time he complained of pain in the chest, saying he thought he had over-exerted himself. He was assisted to his ward and put to lie down. On examination, nothing could be detected beyond weakness of the heart's action. He gradually improved, and was feeling so well this morning that he was anxious to return to work." The explanation of these events, as given by the Medical Superintendent in his report to the coroner, would appear the most satisfactory. "It would seem probable that when exerting himself on the 7th inst., some of the degenerated fibres on the inner aspect of the ventricular wall gave way, and that the walls being thus weakened, a rent was gradually formed under the pressure of the ventricular contractions, the rupture becoming complete immediately before death." I am not able to say if this is a unique case as regards the lapse of time between the first syncopal attack and that which proved fatal, but at least it is one that is quite exceptional.

In reference to these cases I wish to mention another, which, although not one of rupture of the heart, has some bearing on the subject. It is in connection with a heart from a female, aged 58, who died suddenly within two months of admission into Colney Hatch Asylum. The patient had previously been in other asylums since the year 1884. She was admitted February 8th, 1892, with recurrent mania, rambling and incoherent, and of dirty habits. She had cardiac disease, and she told me that she had had rheumatic fever. When seen ten days later she had a wild expression, both eyes were prominent, and the pupils were contracted, especially the right. She also complained of pain at the top of the head. On March 17th, whilst being undressed, she fell back in a faint and died almost instantaneously.

Post-mortem.—The cause of death was found to be meningeal hæmorrhage. The usual thickened and adherent condition of the membranes common to chronic brain disease was present, and in addition a recent thin blood-clot spread over the inner surface of the dura mater on the right side. Situated on the inner side of the dura mater, 2 inches above and 1 inch behind the right external auditory meatus, was a circular thickening, the size of a two-shilling piece, the edges of which were firm and united to the dura, but the central part was soft and breaking down. It gave the impression that it had been a slowly forming hæmatoma, due to the rupture of a vessel in the dura mater, which had suddenly given way, thus

causing the thin clot over that side of the brain, and resulting in the sudden death. But it is to the condition of the heart that I wish to draw attention. There was hypertrophy of the left ventricle, and a considerable increase of fat on the right ventricle and auricle to what exists in health. There were, in addition, minute hæmorrhagic spots on the epicardial coat in several places, but more especially clustered in the neighbourhood of a vessel to the right side of the left ventricle, about 1½ inches from apex; although they existed elsewhere. Microscopically there is seen excessive fat beneath the epicardium, with extravasation of blood-corpuscles and pigment at these spots. There were firm old pericardial adhesions. The aorta was atheromatous and slightly calcareous, as were also the coronary arteries. To complete the account of the *post-mortem* appearances of this case, I may mention that both lungs were firmly adherent at their apices. The upper lobes of both lungs contained vomicæ, but the cavities were dry, and there was calcareous deposit in their walls and adjoining parts, showing phthisis of old standing, possibly quiescent; but there was slight pneumonic consolidation at the base of the right lung. The liver was large, weight 46½ oz., extremely fatty and congested. The gall-bladder contained seven or eight blackish pigmentary calculi. Both kidneys were deeply congested with some adhesion of their capsules, and the left contained several cysts.

In this case we had not the rupture of the wall of the heart, but that of an artery in the dura mater producing death. In this heart, however, we have, I think, just such a one as that in which rupture is likely to, and does, occur. It so happened that here the vessel within the cranium was the first to give way, but it appears to me beyond doubt that, supposing the disease of the arteries had not been in quite so advanced a stage, we should in all probability, within a comparatively short time, have had a further case of rupture of the heart, occurring probably in the left ventricle, where there was excessive ecchymosis in its wall, viz. at the "usual spot."

I will pass over the condition found within the cranium with the remark that its associations with the symptoms noted during life are obvious, but not without interest.

Of the six cases of rupture of the heart previously recorded in the 'Transactions' of this Society, none were from persons of unsound mind. In the two caused by violence the rupture took place in the right ventricle, whereas in the remaining four that

were "spontaneous" it occurred in the left ventricle; in the latter cases the muscular tissue was degenerated. One of these cases (Dr. Hooper's, vol. xix, p. 186) lived for about three days, during which time the patient complained of intense pain in the left side of the chest, and sat "doubled up."

An excessive accumulation of adipose tissue on the heart with a diminution in thickness and degenerated condition of the muscular substance is found not infrequently in the case of aged lunatics, and no doubt accounts for some deaths from heart failure when such are attacked by catarrhal affection of the lungs; but the following case may be mentioned as an instance of sudden death where a heart of this description existed without any lung complication. In 1889 a female patient aged fifty-seven, the subject of chronic mania for seventeen years, and who had suffered during that time from occasional fainting attacks, was suddenly seized with spasm and syncope after eating a hearty meal, and died in a few minutes. At the autopsy some blood-stained fluid was found in the pericardial and in both pleural cavities. The left ventricular wall was exceedingly thin on its anterior aspect for a space the size of half-a-crown, and was here of a dirty yellow colour, but no rupture could be found. The aorta and its branches were very atheromatous. The lungs were healthy. A number of other cases where death has resulted from syncope dependent on "fatty heart" are also on record.

Spontaneous rupture of the heart in the insane is not so rare an event. The following four took place in Colney Hatch.

In 1879 a female patient died suddenly from rupture while suffering from broncho-pneumonia. She was fifty-seven years of age, and the subject of mania of two years' duration. The aperture in the left ventricle was nearly an inch in length, the muscle soft with much fat on the surface of the heart, but the valves were said to be healthy. In this case the liver was hobnail, and the right kidney was filled with calculi.

Another female died in 1884 who, during life, had exhibited many signs of degenerative disease of the vascular system. On admission, two years previously, it was noted that she was of a low type, both mentally and physically, with a livid complexion, dilated capillaries of the face, and varicose veins of the legs, also signs of cardiac disease. She was violent and maniacal, and had drunk heavily. Later on there developed left hæmatoma auris and

ecchymotic swellings about the inner side of the knee and buttock, and gangrene of the right toes supervened. She died suddenly, aged fifty-eight, and a rupture of the left ventricle was found $\frac{3}{4}$ inch in length; the valves were fairly healthy, but the aorta was atheromatous.

In 1886 a woman aged seventy-three, suffering from senile mania for eight years, fainted while having her dinner, and died almost immediately. She had been excessively talkative. A longitudinal rupture, an inch in length, on the outer surface of the left ventricle communicated by a tortuous channel with a transverse rent, $\frac{1}{4}$ inch long, within the ventricle. The aorta and its branches were much calcified, the valves healthy.

In 1888 an old man, seventy-one years of age, who had been an inmate of the workhouse for thirty years, but had become demented, restless, and deluded, was transferred to the asylum, where he only lived a few weeks; he had a faint one morning, and died in a few minutes. In addition to the existence of a rupture of the left ventricle, there was found occlusion of the right vertebral artery within the cranium.

Two cases, occurring in other asylums, were reported in the 'Journal of Mental Science,' January, 1892; one of these was in a man who died instantaneously, the other being a woman who lived forty-eight hours after the first indication was observed.

In all these cases the rupture occurred in the wall of the left ventricle, and the heart was exceedingly fatty, and there was atheroma of the arteries, as well as other degenerative changes present in the abdominal organs. The cause of the rupture is undoubtedly the very diseased condition of the heart, and I do not think we can be surprised at its occasional occurrence, nor do I see how we are to prevent this form of death in our asylums. My own experience so far has been that a diseased condition of the heart and blood-vessels is the rule in patients dying in lunatic asylums, and that it is quite exceptional when making a *post-mortem* to find a heart which may be looked upon as healthy. The same may be said of the arteries, which often appear to be primarily affected, for it is not uncommon for the heart to be greatly hypertrophied without showing any marked valvular change. Combined as this is with chronic interstitial nephritis, it forms an exceedingly common condition, and one on which I may have occasion to remark at another time. *February 7th*, 1893.

2. *Rupture of heart.* (*Card specimen.*)

By NORMAN MOORE, M.D.

THE specimen was sent from Umballa, Punjaub, by Surg.-Capt. T. A. Dixon. The heart is that of a native boy, aged 8 years, who was strangled and thrown into the water. The left ventricle is ruptured near the apex. The muscular tissue is undegenerate. The rupture seems to point to pressure on the chest at the time of strangulation. *February 7th*, 1893.

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3. *A case of fibroid degeneration of the whole of the endocardium of the right ventricle.*

By W. HALE WHITE, M.D.

JOSEPH D—, aged 42, was admitted under my care into Guy's Hospital, February 13th, 1892. He had phthisis at the right apex, also slight albuminuria, which disappeared from time to time, but always came back again. The amount of urea passed was constantly, whatever his diet, considerably below the normal quantity, often less than half. There was ascites, for which he was tapped twenty-seven times, eleven pints being on the average withdrawn at each tapping. The ascitic fluid never contained more than the merest traces of urea. When it was withdrawn, the liver could be felt to be enlarged with a thick margin. The heart was dilated, and there was a faint apex systolic murmur. The pulse was never hard; the eyes were healthy. There was no history of gout, lead, syphilis, or rheumatic fever. We felt sure he had phthisis and chronic peritonitis and perihepatitis, and this turned out to be correct. We did not know how to account for the dilatation of the heart, and the suggestion was made that he had chronic Bright's disease, which would explain the cardiac dilatation, the occasional albuminuria, the diminution of urea, and the chronic peritonitis. This suggestion turned out to be wrong. He died January 6th,

1893. For the last few weeks of his life he was so weak and feeble that he could not be examined.

It was found that the right pleural cavity contained some serum; on the left side the cavity was obliterated by adhesions. There was much phthisis in both lungs, but it was chiefly fibroid, remarkably quiescent, and most of it had healed.

The heart weighed 19 oz. The left side was healthy, the cavities were normal, and the endocardium was not thickened. The right ventricle and auricle were greatly hypertrophied and dilated. On section the muscle was tough, and there was chronic thickening of the endothelial surface of the lower two thirds of the ventricle. This thickening was white and glistening, tough to the knife, and at least ¼ mm. thick. The muscle itself was nowhere fibroid, and to the microscope it appeared healthy. The coronary arteries were slightly thickened. The tricuspid orifice measured 4½ inches in circumference; the flaps were a little shortened, and there was a fringe of minute vegetations along the margin, but the valve was not thickened, and there was no water-marking suggesting any long-continued regurgitation. The circumference of the pulmonary artery was 3¼ inches, and of the aorta 2¾ inches. There was marked atheromatous degeneration of the pulmonary arteries. The left pulmonary artery contained a soft adherent *ante-mortem* clot.

The peritoneal cavity contained five pints of fluid.

The liver weighed 47 oz.; it was nutmegged but not cirrhotic. Its capsule presented a typical instance of universal perihepatitis.

There was much capsulitis of the spleen and a fair amount of chronic thickening of the peritoneum. The structures in the transverse fissure of the liver showed no evidence of obstruction or constriction. Except for one or two old depressed scars the kidneys were normal. *January* 31*st*, 1893.

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4. *Case of disease of the aortic valve and pulmonary tuberculosis.*

By J. Edward Squire, M.D.

The patient, a labourer aged 46, was admitted into the hospital, where he had applied for relief for a cough. He had served fifteen years as a soldier in India, and had there suffered from

malarial fever; since leaving the army, ten years ago, he had been employed as a labourer on the railway. His work had not been excessively heavy.

There was no history of rheumatism nor of syphilis. With the exception of malarial fever in India he had had no illness, and enjoyed good health until the last five years. He had had a dry cough for the past two years. Three months ago he had an anginal attack whilst at work.

On admission there was much cough, with frothy expectoration. The temperature was raised in the evening. Bronchitic râles were abundant in the right lung, with signs of infiltration (congestion) at both bases. The cardiac dulness was increased towards the left; the apex-beat was felt directly below the left nipple. A friction rub was felt over the cardiac area towards the apex. At the apex a prolonged murmur replaced the first sound. At the base two murmurs (systolic and diastolic) were heard, the systolic being distinguished by a difference in tone from that at the apex. There were no signs of aneurysm. The brachial arteries were tortuous, firm, and visibly pulsating.

Four days after admission, dyspnœa and faintness, with sensation of choking, preceded collapse and death.

At the autopsy the heart was found to be enlarged. The mitral orifice was dilated, and the curtains of the valve were puckered, with beaded edges. There was no roughness of the endocardium.

Of the aortic valves, one had its margin roughened; a second had a large oval perforation; and from the free edge of the third a large calcified vegetation, about three quarters of an inch in length, projected upwards into the aorta. The vessel itself was markedly atheromatous, and in some parts of the arch atheromatous ulcers were found.

In the lungs, besides the conditions recognised during life, there was, in the right apex, some undetected tubercle. A scar on the surface of the organ marked the seat of an ovoid caseous mass, about 1½ inches in its longest diameter. This was surrounded by a well-marked fibrous capsule, and outside this was a zone of congestion. Scattered through the upper lobe of this lung were a few much smaller tuberculous nodules, some already breaking down.

The spleen was enlarged in its long diameter to about 9 inches, but not thickened. Its lower end was especially firm and dark-

coloured. The liver was cirrhotic, and the kidneys also fibroid and cystic.

The case is interesting from several points of view. It is curious to find such serious lesions in both heart and lung, with so little inconvenience to the patient until a week or so before death. With the exception of an "attack of giddiness" three months ago, the man had apparently felt no marked inconvenience from his heart.

The causation of his cardiac disease offers a good field for conjecture. There are several circumstances incident to army service, both at home, and more especially abroad, which would favour endocarditis and atheroma.

The right lung presents an interesting example of tubercular disease, advancing to caseation, but then becoming quiescent through limitation of the mischief by fibrous overgrowth round the affected portion of the lung, and being lighted up again later, probably on the escape of some of the imprisoned bacilli.

The tuberculosis of the lung was not diagnosed during life. The temperature chart gave rise to suspicion, but when I examined the chest, the bronchitic râles were sufficiently abundant to mask any auscultatory evidence of the active tuberculous changes found after death.

The presence of the large caseous mass in the apex was not suspected. This mass is worthy of close inspection. The central caseous portion I believe to be much older than the smaller tuberculous nodules observed in other parts of the same lung. When the mischief happened there is little or no evidence in the history to show; probably it was some time in the last five years. The fibrous growth around this shows an attempt at natural cure by "encapsulation." Then we have the recent congestion (inflammation) around. Is this a not yet completed part of the process by which the capsule is formed, or does it indicate the escape of bacilli through the fibrous wall? That bacilli have escaped may be inferred from the recent nodules of still increasing tubercular disease scattered through the upper lobe of this lung. This exemplifies what is very liable to occur after apparent cure of tuberculosis, viz. a re-infection from the quiescent tubercle. The co-existence of tuberculosis of the lung with heart disease is of common occurrence. *May 2nd*, 1893.

5. *Fungating endocarditis of aortic valves in an infant eleven months old.* (*Card specimen.*)

By G. Newton Pitt, M.D.

The heart presents no abnormality beyond the condition of the aortic valves. They are thickened and incompetent. The anterior valve is sloughing, and is in an early stage of fungating endocarditis. There are two aneurysmal pouches in its lower part, which project outwards to the aorta, but they have not perforated. There is a small atheromatous patch above this valve on the aorta, due to the impact of the regurgitant stream.

The remaining viscera were normal, with the exception of some broncho-pneumonia and atelectasis of the base of the right lung.

The specimen is from an infant of eleven months, which was admitted moribund, with a loud basic systolic bruit; and the house physician, Mr. C. Young, also heard a diastolic bruit.

The child had previously been under Dr. Pye-Smith for constipation and supposed congenital heart disease.

This is an earlier age for aortic disease than any cases which I have found recorded in books readily accessible to me; nor have I found any case of fungating endocarditis in so young an infant.

There is no reason to suppose the disease was congenital, but the cause of the fungation remains undetermined; it was evidently very virulent, as the whole of one cusp was softening and commencing to slough.

It is rare to find endocarditis in children under three, although pericarditis is not so infrequent. *January* 31*st*, 1893.

6. *A case of ulcerative endocarditis associated with stenosis of the conus arteriosus and affecting chiefly the pulmonary valve, with ulceration of the main pulmonary artery.*

By J. Jackson Clarke, M.B.

The following abstract of the clinical notes has been kindly furnished by Dr. H. A. Caley, Medical Registrar at St. Mary's Hospital.

A. G—, aged 21, female, admitted to St. Mary's Hospital under Dr. Broadbent on December 16th, 1891, died May 22nd, 1892. The main subjective symptoms were pain in the præcordial region with some dyspnœa; and there was intermittent pyrexia of the most marked type, with frequent rigors during the latter part of the illness. The physical signs were visible pulsation in the second left intercostal space, with marked vibratory systolic thrill, and loud vibratory systolic murmur, most intense over the above-named space, but audible over the entire cardiac area and over the greater part of the chest and back. In the latter part of the illness there was evidence of embolic infarcts in the lungs.

The specimen is in the museum of St. Mary's Hospital (No. 607A). I have described it thus:—"A heart with the great vessels and portions of the trachea and lungs. The base of the infundibulum is so much contracted that it only admitted the tip of a little finger. At this contracted portion the endocardium is slightly thickened, forming a visible white ring. Immediately above this ring is a cluster of soft vegetations. The pulmonary valves have been all but completely destroyed, their remnants being covered with prominent vegetations which, in the fresh state, were quite soft. The walls of the main pulmonary artery present large patches of warty vegetations. The terminations of both right and left pulmonary arteries are blocked by large emboli. The smaller branches of the pulmonary arteries seen on the cut surfaces of the lung are all thrombosed. The pleural surface shows patches of lymph and dark areas which correspond to solid portions of lung. In the recent state these were seen to be infarcts, in some of which purulent softening had begun. The

right ventricle is more than twice its normal thickness. The valves and cavities on the left side of the heart are normal."

Microscopic section showed the fibrinous vegetations to contain, in their superficial portions, masses of streptococci, growing exactly as in pure artificial cultures. *November 1st*, 1892.

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7. *Disease of pulmonary valve.* (*Card specimen.*)

By J. A. Ormerod, M.D.

One cusp was unfortunately cut across in opening the heart. It will be seen, however, that all the cusps of the pulmonary valve are thickened and baggy, and that the line of junction between two of these cusps is so far reduced in dimensions as to suggest at first sight that only two cusps exist. On folding the valve together it appears that some stenosis must have existed during life. The surface of the diseased valve is quite smooth. There is some hypertrophy of right ventricle. Weight of heart 13 oz. The heart is otherwise, except for trivial thickening of the mitral, quite normal.

The patient, a man aged 24, under the care of Dr. Andrew, was the subject of phthisis, and was admitted for hæmoptysis, of which he died before an accurate history could be obtained or a minute examination made of his chest.

A blowing systolic murmur was heard at the base of the heart, propagated over a considerable area in all directions, and thought to be hæmic. He was cyanosed. *November 1st*, 1892.

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8. *Intra-auricular cardiac polyp.*

By A. F. Voelcker, M.D.

The specimen was obtained from a woman, aged 53, who was admitted into Middlesex Hospital in March, 1892, under the care of Dr. Kingston Fowler.

The patient had had good health up till the Christmas of 1891, when she developed a cough which she attributed to exposure to fog. She took to her bed, and remained there till her admission into Middlesex Hospital on March 8th, 1892. About the middle of February her legs began to swell and her breathing became more difficult. There was no family history of rheumatism, and the only previous illness was an attack of smallpox at twelve years of age. On admission she was cyanosed, and had considerable œdema of the lower limbs and trunk, and extensive general bronchitis. The pulse was weak and irregular, the veins of the neck filling from below. The heart's apex-beat was in the fifth space in the nipple line, but the heart sounds were obscured by loud rhonchi. On March 10th she was worse. A systolic murmur was heard plainly at the apex, and the notes state that "the cardiac rhythm is suggestive of a presystolic murmur, but it cannot be definitely defined." Patient gradually got worse, and died on March 12th.

At the autopsy the various organs showed signs of chronic venous congestion. The liver presented a well-marked nutmeg appearance, the spleen was enlarged and firm, and the kidneys were cyanotic. The lungs were emphysematous, and there was extensive bronchitis. No infarcts were found in any of the organs.

The heart weighed 17 oz. and was considerably enlarged, especially on the right side, the walls of which were pale, and contained 12 oz. of dark clot. The tricuspid orifice was considerably dilated, and the cusps showed chronic thickening at the edges, but no vegetations. The pulmonary valves were natural. The right auricle was much dilated, but there was no adherent

clot in the auricular appendix. A probe could be passed obliquely through the foramen ovale into the left auricle. The left ventricle was somewhat hypertrophied and dilated. The mitral orifice admitted two fingers, and the cusps were somewhat opaque; but no vegetations were seen. The aortic valves were natural. On opening the left auricle there was seen projecting into its cavity a yellow gelatinous-looking mass, of the shape and size of a champagne cork, attached by a broad base to the lower part of the position of the foramen ovale. At its attachment the mass was paler, and was streaked with yellow lines extending from the septal wall of the auricle. The surface of the mass was smooth, and presented a few yellow spots and red streaks. The mass was seen to fit over and into the mitral orifice, and appeared to have been moulded into shape by it. The specimen, now in the museum of Middlesex Hospital, gives a very imperfect idea of the size or appearance of the mass before it had shrunk and lost its gelatinous appearance as a result of the action of the spirit used in its preservation.

A slice of the mass was removed from its lower surface, and sections were made and examined with the microscope.

The mass is seen to be composed of structureless laminated layers of fibrin enclosing small round cells, blood-pigment, larger oval cells, and embryonic vessels. The laminated appearance is most marked on the surface, and the numerous cells enclosed between the layers of fibrin give at first the impression that they are the nuclei of spindle-shaped cells; but in places the cells are seen forming columns, and forming the embryonic vessels seen in other deeper parts of the section. I am unable to make out any layer of endothelium over the surface of the mass. In the deeper parts the ground substance has not the laminated appearance before noted, and the fibrin seems more hyaline and swollen, and there are numerous larger oval cells, many containing pigment granules in their interior. Numerous vessels are seen with a well-marked endothelial lining of oval cells, but no muscular coat, and in the lumen of some of the larger vessels only a granular *débris* and no actual red corpuscles can be seen, though blood-pigment granules and small round cells are seen in some, while in others red corpuscles can be recognised. The specimen (Fig. 1) shows the stages of development of the embryonic vessels from the solid columns of cells to the definite vessels—a condition described by Prof. Delépine as one of potential vascularisation.

The specimen is interesting on account of the unusual position of this cardiac polyp, and on account of the distinct evidence we have of a process of vascularisation of the mass.

FIG. 1.

Section of organising cardiac thrombus. At the surface laminated blood-clot with columns of cells between the laminæ; the deeper part of the clot not laminated, traversed by embryonic vessels. Zeis. Obj. A.A. Oc. 4.

Prof. Delépine, in a valuable paper read before the Pathological Society in January, 1890, "On a Description of a Cardiolith," vol. xli, 'Trans. Path. Soc.,' has collected the literature of the subject. Allan Burns, in 1809, refers to a cardiac polyp in which, by forcing air into the coronary artery, some few vessels in the new-formed substance could be inflated. Rokitansky mentions that he has never been able to detect vessels in cardiac polypi. Walshe mentions two varieties of polyp, one of which is more or less markedly vascularised. In Prof. Delépine's case of a cardiolith

well-formed fibrous tissue, and only a few spindle-cells; there are only occasional giant-cells. There is no definite caseation. The small arteries in the neighbourhood of these granulomata are healthy.

From a man aged 34, who died with very extensive fracture of the skull, as the result of a fall off a house.

There were scars in the groin and on the glans penis. The testes weighed 6 oz., both being almost transformed into fibrous tissue; there was a small gumma in the left. The tunicæ vaginales were universally adherent on both sides and much thickened.

There were no gummata in the liver. The lungs were normal. The arteries at the base of the brain were healthy.

The points of interest are the very early condition of the future gummata, and the fact that, being multiple, they are almost confined to the wall of the right ventricle and to the septum ventriculorum.

January 17th, 1893.

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10. *Congenital malformation of the heart.* (*Card specimen.*)

By Arthur F. Voelcker, M.D.

The heart is considerably enlarged, both ventricles being about the same thickness. It presents the following abnormalities:— The interventricular septum is incomplete, and the aorta rises from both ventricles; the pulmonary artery is small, and the valves are adherent, leaving only a small chink, through which a green rod has been passed; the margins of the chink are calcareous. The aorta gives off from the concavity of the arch three arteries to the lungs, and the ductus arteriosus, which is occluded below; the foramen ovale is patent; the tricuspid valves show numerous vegetations, and the aortic cusps a few.

From a female child, aged $7\frac{1}{12}$ years, who had been an out-patient under Dr. Abercrombie at the Hospital for Sick Children, Great Ormond Street.

She was a full-term child. No family history of rheumatism. Pregnancy normal.

When first seen, in July, 1889, she had cold extremities and cyanosis, with clubbing of fingers. The heart's position was natural; a rasping murmur was heard at left base and over the centre of the heart; it was not heard behind.

In October, 1891, she was much cyanosed, with extreme clubbing of fingers; feels very cold in winter. The systolic murmur was loudest over third left costal cartilage.

September, 1892.—No material change was notable.

January 21st, 1893.—Had been as usual. Was out in her "mail cart," and felt queer in her head and died in a few minutes.

Post-mortem.—Cyanosed; marked clubbing; venous engorgement of internal viscera; one old infarct in lung. Heart as described above. (Head not examined.) *February 7th*, 1893.

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11. *Spontaneous rupture of inner and middle coats of aorta; leakage into pericardium.*

By H. D. ROLLESTON, M.D.

ABOUT a third of an inch above the junction of the right anterior and of the posterior segments of the aortic valves there is a complete rupture of the internal and middle coats, which runs transversely, with a slight inclination upwards and to the left, for an inch and a half.

The external coat and the visceral layer of the pericardium are almost completely intact, and are slightly bulged outwards into the cavity of the pericardium. Together they form a thin almost transparent membrane, with a small hole, the size of a pin's head, near the centre. The inner and middle coats have separated from the external coat for about half an inch in the transverse plane, but there is no dissecting aneurysm. The aorta shows diffuse atheroma, which predisposed to the rupture, and the arch of the aorta is slightly dilated, but not sufficiently to be called a fusiform aneurysm. At the situation where the ductus arteriosus joins the aorta the artery becomes rather suddenly of its normal calibre. The pouch projects to the right side of the ascending part of the arch of the

aorta; on its outer surface a small piece of clot was found at the *post-mortem*, marking the situation of the leaking point.

At the autopsy the pericardium was found to contain 24 oz. of red jelly-like clot and a considerable quantity of serum; this quantity of blood must have been gradually driven through the small hole in the external coat, and thus have stretched the pericardium to the extent that it contained more than 24 oz. instead of about 7 oz., which is the quantity usually found when an aneurysm bursts freely into the pericardium and produces sudden death.

This specimen was removed from the body of a cabman, aged 44, who fell off his cab dead, and was brought into St. George's Hospital. The cab was in the cab rank at the time, and there is no evidence as to the suddenness of the event, except that there was a belladonna plaster over the heart, as if he had had some pain. The rupture of the coats of the aorta appears to have been spontaneous. It is to be noticed that there is no dissecting aneurysm; as might, perhaps, have been expected. The blood bulged the external coat out into the cavity of the pericardium, and separated it just for this small area from the inner and middle coats. The explanation is probably that the blood-pressure was exerted in driving the blood through the small hole in the outer coat into the pericardial sac, and not in separating the coats.

There are a considerable number of cases of pin-hole rupture of intra-pericardiac aneurysms, with large quantities of blood in the pericardial sac, recorded in the 'Transactions' of this Society, but I have not found one in which this gradual filtration of blood resulted from what appeared to be a spontaneous rupture of the two inner coats. *December 20th*, 1892.

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12. *Traumatic dissecting aneurysms of the aorta.*

By H. D. Rolleston, M.D.

There is some atheroma at the base of the aorta. Just above the aortic valves there is a transverse rupture, an inch and a half long, passing abruptly through the inner and middle coats. Extending distally from this point there is a dissecting

aneurysm between the external and middle coats of the aorta. This fresh channel does not surround the whole circumference of the arch of the aorta, but passes first to the right of the ascending part, then behind the arch, and opens again into the aorta on the inner bend of the arch, close to the scar due to obliteration of the ductus arteriosus.

There is thus a channel containing blood between the middle and outer coats of the aorta. A probe can be easily passed from one opening to the other. There is jelly-like clot in the dissecting aneurysm; the lumen of the aorta is much narrowed by the inward projection of its coats by the dissecting aneurysm.

There is no continuation of the dissecting aneurysm on to the vessels arising from the arch of the aorta.

Nine inches from the aortic valves the inner and middle coats of the thoracic aorta are again separated from the adventitia, and the blood has passed in between them and bulged the detached inner and middle coats into the lumen of the aorta. This second dissecting aneurysm does not appear to communicate with that in the arch of the aorta.

At the *post-mortem* the right sixth rib was fractured an inch and a half from its junction with the costal cartilage; there was a transverse fracture without any displacement of the sternum at the base of the fifth ribs.

There was a large mass of decolourised blood-clot in the substance of the left lobe of the liver near the falciform ligament, without any rupture of the peritoneal coat.

There was a large hæmorrhage into the medulla of the right supra-renal capsule.

Kidneys normal.

Lungs: right, 32 oz.; left, 26 oz.; both softened, decomposed, and œdematous; no pneumonic consolidation.

Pericardium normal; no effusion.

Heart: left ventricle dilated and hypertrophied; valves healthy.

There was no fracture of the spine. The brain was normal.

Clinical history.—The patient was a stableman, 45 years of age, and in fair health. While at work on April 18th, 1873, at 8 a.m. he received a kick from a horse in the front of his body, causing a good deal of pain. He came to St. George's Hospital, and was admitted under Dr. Dickinson at 1.30 p.m.; his condition then was as follows:—Bloated though pallid, sweating heavily,

complaining of pain at the place kicked, restless and short of breath, legs slightly œdematous, pulse extraordinarily hard, 116. Urine slightly albuminous. Heart acting violently, apex in sixth space, two inches beyond the nipple line, with a systolic mitral murmur, a systolic aortic, and a very loud and extensively audible musical diastolic murmur. Temp. 100°.

Extreme dyspnœa rapidly came on with some blood-stained expectoration, the lungs being fully resonant and loaded with sonorous sounds. Respirations 48. It seemed that his condition was due to uræmic asthma, or to some laceration of the lung not discovered by a necessarily short examination.

At 2.30 p.m. he was bled to 14 oz.; this gave obvious relief at once, and undoubtedly saved him from immediate death. An hour or so later the bandaged slipped, and more blood (3 or 4 oz.) escaped.

Next day, April 19th, the relief given by the loss of blood was still in effect, though the man's condition was far from favorable. Profuse sweating was very marked, and the breathing remained rapid (44). Pulse 108, tense, but less so than before. He did not sleep, and on April 20th he showed much tremor and busy delirium. During that night he slept, and on the 21st was more rational, though not at all improved as regards his dyspnœa. Respirations 56. Now was found what had not been present before, a strong thrill over the aortic cartilage, synchronous with the booming diastolic murmur.

The dyspnœa increased; the temperature rose to 102·4°; and on April 22nd he died.

For the clinical notes I am indebted to Dr. Lee Dickinson, Medical Registrar to St. George's Hospital.

Remarks.—The dissecting aneurysms were apparently recent, and, as shown by the history of the case, developed after the injury was received, and probably were directly due to it, the internal and middle coats being, it may be supposed, ruptured at the time of the injury, and the dissecting aneurysm developed subsequently.

In a case shown earlier in the session before this Society there was a spontaneous rupture of the internal and middle coats of the aorta in the same position as in this instance. There, however, was no dissecting aneurysm. This was probably due to the fact that the external coat had a small hole in it which allowed the

blood to pass gradually into the pericardium, and thus diverted the blood-pressure from dissecting up the arterial coats.

The kick from the horse appears to have struck the patient in the present instance to the right of the middle line at the level of the fifth and sixth ribs, the force being conducted in two directions,—upwards along the sternum to the arch of the aorta, where it produced rupture of the two inner coats; and also backwards, giving rise to a second dissecting aneurysm in the thoracic aorta and to hæmorrhage into the substance of both the liver and the right supra-renal without rupture of their surfaces. The high tension of the pulse and the extreme dyspnœa, possibly due to injury of the cardiac and other plexuses of the sympathetic around the aorta, and the great relief obtained by venesection, are points of interest.

In the forty-second volume of the Society's 'Transactions'[1] Mr. Bowlby has recorded a case which is somewhat comparable to this. The first rib and the clavicle were fractured, and the subclavian artery, at the junction of the first and second parts, had its internal and middle coats sharply cut across as if by a ligature. The coats had then been dissected off for a distance of more than an inch, and had met and thus blocked up the lumen of the third part of the artery. Here, as the result of traumatism, a dissection of the internal and middle coats has been effected by the blood-stream as in this instance. *May 16th*, 1893.

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13. *Aortitis.* (*Card specimen.*)

By H. D. Rolleston, M.D.

The whole of the intra-thoracic aorta is affected; as it passes through the pillars of the diaphragm the coats of the vessel become normal. In the affected part the intima is greatly thickened, and appears somewhat translucent; on transverse section the thickened intima is seen to be thrown into folds. This change is very diffuse, but does not spread into any of the branches of the

[1] P. 79.

aorta, though it narrows the origins of the left coronary and of the left carotid artery.

In the arch of the aorta there are patches of ordinary atheroma, undergoing degenerative changes.

Microscopically the changes of chronic endarteritis were seen in an extreme degree, but there was no evidence pointing definitely to syphilis as a factor.

At the *post-mortem* the aorta was more than normally adherent to the tissues around.

From a man aged 44, who died suddenly. There was an old scar on the penis, and a small fibroid patch in the left testis.

A somewhat similar specimen was shown earlier in the session by Mr. Beadles. *May 16th*, 1893.

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14. *Aneurysms of the vertebral and internal carotid arteries.* (*Card specimen.*)

By William Henry Battle.

Situated on the left vertebral artery, and arising from the portion between the second and third cervical vertebræ, is an aneurysm of the size of a nutmeg. Part of the wall of this is absent, and shows the interior of the sac, containing a small amount of clot; most of the clot has, however, been removed to show the two openings in the artery communicating with the interior of the sac. The wall of the sac is very thin, and had given way during life. It extends from the level of the fourth cervical vertebra to the lower border of the transverse process of the atlas. About a quarter of an inch of the posterior wall of the artery is exposed in the sac, and extends between the two openings of the artery.

A quarter of an inch above the bifurcation of the common carotid, the internal carotid of the left side shows a somewhat fusiform aneurysmal dilatation. A silk ligature encircles and occludes the common carotid an inch below the bifurcation.

From a child (male) aged 2½ years, admitted to the Royal Free Hospital on June 25th, 1892, with diaphysitis of right femur.

Abscess incised. On July 31st abscess on left side of neck incised. On August 23rd Mr. Battle was first requested to see the child, in consequence of repeated arterial hæmorrhage from the neck. This was associated with high fever. The opening in the neck was enlarged, and a soft pulsating mass found extending up to the base of the skull. As there appeared to be some diminution of pulsation on pressure over the common carotid, it was supposed that the aneurysm was in relationship with the internal or external carotid; but as it quite obscured these vessels, and there was no time to be lost, a ligature was placed on the common carotid. The child died next day, with high temperature.

May 16th, 1893.

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15. *Syphilitic (?) disease of aorta, with dilatation of the heart and occlusion of the left subclavian artery.* (*Card specimen.*)

By CECIL F. BEADLES.

THE specimen is that of an aorta and heart.

The arch of the aorta and the upper part of the descending aorta are much dilated. The wall is greatly thickened throughout the entire thoracic portion, its inner lining being very uneven and rough. It is very tough, with no calcareous deposits. This condition ends sharply about two inches above the cœliac axis (viz. where the vessel passes through the diaphragm), below which point the wall of the aorta is of normal thickness, and the inner coat is smooth and shining and apparently normal, as are also the iliac arteries.

The commencement of the left subclavian artery is small, and its orifice is completely occluded by a plug of firm fibrous-like tissue, which is firmly adherent to the wall of the vessel. This is apparently an organised clot, but how far it extended along the artery I am unable to say. It does not project into the aorta. The other branches arising from the arch of the aorta, viz. innominate and carotid, are somewhat small at their orifices, but the vessels beyond appear quite healthy. The openings of the intercostal arteries are

almost obliterated. The pulmonary artery is large, but its coats are healthy, as are also the veins leading into the heart.

The heart is considerably dilated. In the recent state before opening it weighed (with arch of aorta) over 40 oz.; after the clots had been turned out the weight was 30 oz. Large black clots were present in all four cavities, with pale clot attached to the columnæ carneæ. There is great dilatation of all four cavities of the heart, without any hypertrophy to speak of. The columnæ carneæ of both ventricles are large and coarse, and the muscular coat towards both apices is very thin; but that of the right auricle is most markedly so, being quite translucent in many places. All the valves are correspondingly large, and they all appear to be incompetent. The mitral admits more than three fingers. The cusps of all the valves are healthy in appearance, except those of the aortic valve, which are thickened. Both the foramen ovale and ductus arteriosus are closed.

History.—Very little is known of the subject from whom the specimen was obtained beyond the fact that he was a man aged 40, who, in November, 1889, had his tongue removed at St. Mary's Hospital, for cancer, which had been noticed only one month. The disease returned in the following February, from which time he had gradually increasing difficulty in opening his mouth. He died suddenly November 3rd, 1890, a few hours after coming under observation. I have been informed that while the patient was in St. Mary's Hospital he showed no symptoms of heart disease.

Post-mortem.—Body well nourished. Mouth could not be opened, owing to destruction and dislocation of the right condyle of the lower jaw. Both the sides and floor of the mouth were extensively ulcerated, and infiltrated with new growth, and dirty fœtid pus. The tongue was entirely absent. The ulceration extended back and involved the upper extremity of the larynx, and the sides of the pharynx. The lymphatic and submaxillary glands beneath the lower jaw were suppurating. The larynx was intensely congested at the upper part, with ulceration of the arytænoid cartilages and of the fold of membrane.

Besides the condition of the heart and aorta already noted, the internal organs presented the following appearances:—There were several ounces of clear fluid in the pericardial cavity, but no adhesions, and about five ounces of serous fluid in both pleural cavities. The lungs were emphysematous.

The peritoneum contained a small quantity of fluid. The stomach was much dilated, and the mucous membrane in a state of venous congestion. The pylorus was narrow. The colon throughout, and the sigmoid flexure, were very contracted and empty. The small intestine was of about normal size, and contained flatus. The liver weighed 70 oz., and was of a typical nutmeg appearance throughout. Spleen small and pale. A small accessory spleen three quarters of an inch in diameter was present, and in contact with the lower end and to the outer side of the left kidney. There was nothing abnormal about the kidneys, but the bladder was contracted and slightly hypertrophied, and contained ammoniacal urine. *February 7th*, 1893.

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16. *Aneurysm of ductus arteriosus and atheroma of pulmonary artery.*

By R. G. Hebb, M.D.

A male, aged 40, was admitted into Westminster Hospital, under Dr. Donkin, for dyspnœa and hæmoptysis. He was a general labourer; had suffered from winter cough for past seven years, and had occasionally spat up blood for last five or six years. He had sciatica five years ago, but no other illness. He had always lived well. No history of alcohol. The shortness of breath came on twelve months ago, and for the past six months he had been losing flesh and quite unable to work. On admission it was found that there was impaired resonance over left thorax in front; a systolic bruit was audible all over left chest, but loudest over area of pulmonary artery, and a thrill could be felt in second left interspace, where also was a visible and palpable impulse. The apex-beat was most marked in fourth left interspace, but could be felt over a wider area. Some crepitations were heard over left lung in front. The physical signs remained very much the same during his stay in hospital, and he continued to spit blood from time to time. He was under observation as an out-patient till his death, and continued to spit blood to the last. The treatment was by potassium iodide and rest. He died outside, and was brought in for *post-mortem* examination.

The body was emaciated. The heart was enlarged, and with aortic arch and main pulmonary artery weighed 18 oz. There was dilatation and hypertrophy of right heart; cavities filled with soft black clot in diastole; endocardium normal and valves competent; aortic arch very atheromatous, somewhat dilated, and exhibiting some slight aneurysmal bulgings. Just beyond and opposite the subclavian is a circular aperture, one eighth of an inch in diameter, leading into a spheroidal aneurysm about the size of a small walnut. It is entirely filled with firm laminated brown fibrinous clot. It abuts against the left bronchus and pulmonary artery.

The main pulmonary, as far as its bifurcation, is thickened, atheromatous, and dilated. The dilatation, which is aneurysmal in character, is about the size of a turkey's egg, and has only one exit aperture, viz. that to its right branch, and this is narrowed down to the size of a goose-quill. The left branch is quite occluded, the site of its original aperture being marked by a furrow. The stump of the left branch is about one third of an inch thick, and the thickening is chiefly due to a concentric fibrosis of its wall, which has obliterated the lumen.

In shape the aneurysmal dilatation is conical, with the orifice of the right pulmonary at the apex, and around this ring the wall is thick and rough, the thickest part being at its outer aspect, where lies the stump of the left branch.

Beyond the aortic arch and beyond the pulmonary aneurysm these vessels and their branches present nothing abnormal to the naked eye.

The left pleura was universally adherent, and the left lung is a mass of pigmented fibroid tubercles; their aggregation is extremely dense in the upper half, and though they are less frequent they extend to the base.

There are only a few fibroid tubercles in the right lung.

In the small intestine are some (15) circular tubercular ulcers. Thyroid large and colloid. Other parts and viscera not noteworthy.

Microscopical examination of the lung shows that it is a good example of fibroid and pigmented quiescent tubercle. I did not find bacilli. In some giant-cells were well-marked vacuoles. The sections also showed a few collections of Russell's bodies and the usual large number of red staining granules seen in tuberculosis.

I think it is probable that the aortic aneurysm is due to im-

perfect obliteration of the ductus arteriosus, and this view is supported by its position and the character of the wall of the sac, which is thin, fibrous, and smooth internally.

The other interesting points are the atheromatous and aneurysmal condition of the main pulmonary artery, the complete occlusion of the left branch, and the conversion of the left lung into a mass of pigmented fibroid tubercles, owing to the deficient blood-supply.

The aneurysm of the ductus arteriosus merits less the term aneurysm, than dilatation of a partially persistent fœtal vessel; while the condition of the main pulmonary artery, its dilatation and the degeneration of its walls, and the pulsation and the systolic bruit during life, suggest that the term aneurysm would be quite as applicable as that I have adopted. *April 18th*, 1893.

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17. *Embolic aneurysm of the pulmonary artery; infective aortic valvulitis, aortitis, and pulmonary endarteritis; patent ductus arteriosus.* (*Card specimen.*)

By PERCY KIDD, M.D.

AT the apex of the lower lobe of the left lung there is a sacculated aneurysm of the size of a shelled walnut, with thick walls, springing from a secondary branch of the pulmonary artery. The sac is filled with laminated thrombus, but the corresponding artery is not obstructed. The aneurysm is not situated in a pre-formed pulmonary cavity, but has developed in the midst of healthy lung tissue.

Heart (weight 22 oz.) shows dilatation and hypertrophy of both sides, more especially of the left. The aortic valves are incompetent and only slightly thickened, the orifice being obstructed by large, soft, fibrinous vegetations. Mitral and tricuspid valves healthy. The pulmonary valves are healthy, but just beyond the valves, on the posterior aspect of the pulmonary artery, there is a crop of warty, fibrinous vegetations attached to the vascular wall.

In the situation of the ductus arteriosus there is a small oval

opening the size of a goose-quill in the left branch of the pulmonary artery, which here communicates freely with the aorta. The edges of this opening were fringed with soft fibrinous vegetations. Just to the right and above this point there is a small sacculated aneurysm of the aorta of the size of a small marble, lying to the left and in front of the trachea. Flat translucent elevations of the aortic wall (aortitis) surround the neck of the sac.

Other lesions present in the case were infarcts in the spleen and kidneys, acute nephritis, and scanty tubercular nodules in the lungs of small size, some softening to form minute cavities.

The organs are those of a single woman, Harriet P—, aged 22 years. The patient had never suffered from rheumatism, but gave a history of dyspnœa and wasting of about twelve months' duration. The physical signs during life indicated great enlargement of the heart, with double aortic, double pulmonary, and systolic mitral murmurs. Signs of pulmonary phthisis were also detected, and tubercle bacilli were found in the sputum.

October 18*th*, 1892.

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18. *Thrombosis of the pulmonary arteries without hæmorrhagic infarcts.*

By G. Newton Pitt, M D.

I have brought before the Society this evening the left lobes of two lungs, the main pulmonary arteries of which are occluded, but neither of them in their pulmonary tissue present any hæmorrhages nor any lesions which would suggest blockage of the main arterial trunk.

Litten and others have shown that the nutrition of the lung is fully maintained by the collateral supply of blood from the bronchial, mediastinal, and pericardial arteries, when the pulmonary artery is occluded by a non-septic clot or foreign body.

(A.) From a man aged 32, who was under the care of Dr. Hale White with chronic phthisis and chronic peritonitis with effusion.

The left pulmonary main artery was completely filled by a

thrombus which adhered over part of the circumference. The thrombus was softening in the centre, and the clotting extended into some of the smaller branches.

The corresponding portion of the lung, which was the part which was least diseased, was free from any extravasation of blood or solid infarction, and presented no abnormal appearances.

(B.) From a boy, aged 5, under Mr. Durham's care, who had an enormous sarcoma of the kidney with thrombosis and growth in the renal vein.

The left pulmonary artery was completely occluded by a soft mass which, microscopically, was sarcoma.

There was some collapse of the bases of both the lungs, and at the posterior surface there was some pneumonic consolidation.

There is no extravasation of blood nor any appearance in the lung to suggest that the left pulmonary artery was completely blocked.

The impression which is gathered from a perusal of almost any of the standard books upon medicine or pathology is, that if a branch of the pulmonary artery be occluded by a clot, whether this be formed locally as a thrombus or carried thither from the right side of the heart or from a vein, the result in most, if not in all instances, will be to produce in the lung tissue, within a short time, an extravasation of blood, which will coagulate and form a solid, wedge-shaped mass, extending up to the surface of the lung and varying in size according to the branch which is blocked. Yet an examination of a number of cases entirely fails to bear out this view.

On the other hand, Gravitz and others have shown that when the pulmonary arteries are experimentally occluded, it is only occasionally that hæmorrhages occur.

Out of forty-six cases of which I have notes from our post-mortem records in which thrombosis had taken place in one or more branches of the pulmonary artery, in only eighteen, or but little more than one third of the cases, was there an extravasation of blood into the pulmonary tissue, and out of twenty cases of embolism, in only six (or, again, about the same proportion) did blood exude. Hence a hæmorrhagic infarction is far from being the usual result of a blockage of a pulmonary artery.

Also it should be noted that of twenty-eight cases in which

the lungs were free from infarctions, the main trunk of one lung was blocked in one half (sixteen) of the cases, and in the rest one or more small branches.

Of the eighteen cases in which there were infarctions, in only three was the main artery blocked and in each there were numerous distal small infarctions with, in one of the cases, small thrombi in the distal hæmorrhagic areas. I would, therefore, insist that when a main trunk is involved it is very unlikely (one in six) that an extravasation of blood will take place, and that when it does occur, instead of one large infarction, several small infarctions will form.

When the smaller trunks are blocked infarction occurs in about one-half (fifteen out of thirty-one).

Cohnheim and others state that thrombosis in the pulmonary arteries is extremely rare, and that when clots are found they are almost always embolic.

Out of sixty-seven cases, in only twenty-one was there definite evidence, at the inspection, that this was the case.

Out of the remaining forty-six cases, in ten only was a thrombus found elsewhere, either in the right side of the heart or in a vein, which might have formed a source for an embolus. In none of them did the evidence appear clear that they were embolic.

I would, at the same time, allow that occasionally a thrombus may be loosened from a vein or the heart and, being carried on, leave no trace of its seat of formation.

In the remaining twenty-nine cases, no such clots in vessels other than the pulmonary were found, and it is improbable that it should be the exception to find them, if, as authors have stated, clotting in vessels outside the lung setting up embolism was the rule.

Moreover, the general associated conditions were such as might readily induce pulmonary thrombosis, which I believe is more common in these arteries than in any other vessel in the body.

Out of 3218 inspections during the last seven years at Guy's Hospital, the relative frequency of thrombosis is as follows :—

Veins.			
Iliac	34	times.	
Femoral	33	„	
Prostatic	27	„	
Internal jugular	17	„	
Lateral sinus	15	„	
Uterine	12	times.	
Inferior vena cava	12	„	
Innominate	9	„	
Renal	8	„	
Pelvic	8	„	
Portal	7	„	

Broad ligament	6 times.
Hepatic, longitudinal sinus, and superior mesenteric, each	4 „
Popliteal, cerebral, and subclavian, each	3 „
In several other vessels clots were noted only once or twice in this period.	

Arteries.	
Pulmonary	40 times.
Aorta	12 „
Iliac	3 „
Heart.	
Right ventricle	23 „
Left ventricle	26 „
Right auricle	22 „
Left auricle	20 „

Besides these forty cases entered as thrombosis in the pulmonary artery, there were twenty-six entered as embolism. In many of these cases the only evidence for calling the clots emboli was the belief that this was the orthodox view to take, but I would urge that all the evidence tends to show that many of the clots had formed initially in the pulmonary arteries, and should be labelled thrombi.

Out of twenty-eight cases in which there were thrombi without infarcts, there was phthisis in seven, malignant disease in six, profound anæmia in three, suppurative peritonitis in three, and the remaining cases were all in feeble patients.

Now, these are all conditions which lead to great enfeeblement, to a very defective circulation, and would be considered a sufficient explanation of thrombosis if the vessel were not the pulmonary: then why should these conditions not be sufficient to induce thrombosis in the pulmonary arteries? We know that such patients are bedridden, breathe very feebly, and therefore have a very slow circulation at the bases of their lungs, and it does seem unreasonable to assume that more than a few, if any, of the clots in these cases are embolic in origin, unless there is further evidence of it. In not one single case, considered as thrombosis, was the clot folded, nor did it present evidence of fracture; in all, the clot fitted the vessel and did not, so far as the reports go, give any evidence of a central embolus with a secondary thrombosis. In some cases, however, probably the circulation may not have been completely blocked.

I think that in future if the appearance of the clot itself in the pulmonary artery is such as to suggest that clotting has taken place locally, it should be considered sufficient evidence upon which it may be labelled a thrombus, and that the impression

that such clots must almost necessarily be embolic in origin is really untenable.

I do not propose dealing to-night at all with the causation of pulmonary apoplexies, but in order to guard against misapprehension, I would state that of course the arteries in the middle of a pulmonary hæmorrhagic clot are blocked; but I have not considered such cases to-night under pulmonary thrombi, but only those in which the artery leading up to the infarcted area is blocked on the proximal side of the hæmorrhage.

I feel justified in this exclusion because, while on the one hand blockage of an artery does not generally produce a hæmorrhage, so, on the other, it can be shown that when hæmorrhages are found, they are not due to blockage of medium-sized arteries, but occur in quite a different set of conditions from those which induce thrombosis.

As in so many other cases, our difficulties are intensified by our nomenclature. The terms pulmonary apoplexy and pulmonary infarction are largely responsible for the confusion.

We require distinctive terms for the hard, wedge-shaped hæmorrhages which generally are not infarctions, and owe their shape and size to vigorous hæmorrhage into pulmonary tissue; also, secondly, for the smaller, softer, nodular hæmorrhages which, if they continue to bleed, would lead to the first form; thirdly, the plum-pudding hæmorrhages due to blood sucked down the bronchi.

If this Society could adopt any suitable nomenclature for such appearances it would be of inestimable advantage.

The propositions which I wish to lay before the Society are:—

1. That the blockage of the smaller pulmonary arteries, and still more frequently of the main branches, often take place without extravasation of blood into the pulmonary tissue taking place.

2. That the clots in the pulmonary arteries, which some authors consider to be almost invariably embolic in origin, are, on a review of a number of cases, found to be more frequently due to thrombosis. At any rate, that in a large number of cases there is not a particle of evidence that they are embolic in origin, while the associated conditions are such as would be accepted as sufficient to produce thrombosis in vessels elsewhere.

3. That thrombosis in the pulmonary artery, so far from being very rare, possibly occurs more frequently than in any other vein or artery in the body. *January 17th*, 1893.

19. *Arterio-venous aneurysm connected with the iliac vessels at their origin.*

By G. Newton Pitt, M.D.

The specimen shown consists of an aneurysmal sac formed by a pouching of the anterior and inferior wall of the left iliac vein for half an inch shortly below its origin. The termination of the abdominal aorta between the iliac arteries opens by an orifice, ¼ inch across, into the sac, which is 1¾ inches long, 1¼ inches broad, and 1 inch deep. The right iliac artery is in front and adherent to its wall for nearly two inches; the left artery lies entirely to the left of the sac. The right iliac vein, which is normal and does not communicate with the sac, lies behind it and runs in its postetior wall for 1½ inches.

The non-dilated proximal half-inch of the left vein opens into the right upper corner of the sac, and the distal part opens freely from the left upper corner, as a tube dilated to a diameter of ¾ of an inch, soon narrowing down as the external iliac vein to the normal diameter of less than ⅜ of an inch. The other changes found at the inspection were as follows:

The whole sac lay in front of the upper end of the sacrum embedded in dense fibrous tissue, which rendered the dissection of the sac a tedious and difficult process.

The left iliac vein and the sac were free from any ante-mortem clot, but in the valves of the left external iliac there were small ante-mortem clots, and some were also found in the left superficial, epigastric, and iliac veins. The sac therefore lay between the right iliac artery and vein, was formed by a pouching of the inferior and anterior part of the left iliac vein, and opened into the aorta, with which it came into contact, between the iliac arteries.

The inferior vena cava is very slightly dilated in its lower part.

The interior of the sac is smooth; the wall is so embedded in fibrous tissue that it is difficult to determine its thickness, but probably it is not more than 1 mm.

There were 8 oz. of fluid in the right pleura.

The heart weighed 6 oz., the normal weight for a child of five

FIG. 2.

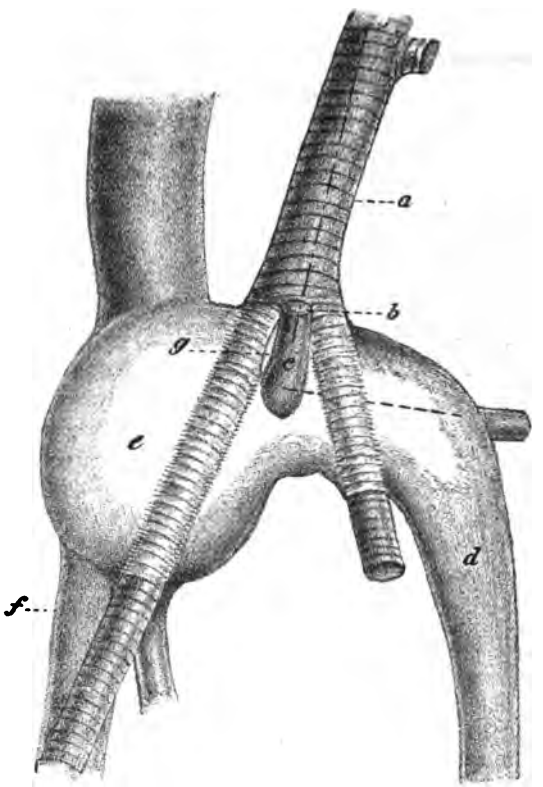

Diagram of anterior surface of specimen.

a. Aorta. *b*. Orifice of communication with sac. *c*. Window cut in wall of sac. *d*. Dilated distal part of left iliac vein. *e*. Sac. *f*. Normal right iliac vein lying behind sac and unconnected with it. *g*. Aperture of communication between sac and undilated proximal part of left iliac vein.

being 2·3 oz. Both sides of the heart were dilated and hypertrophied, but there was no valvular disease.

The pericardium was healthy, and a triple sound which had suggested pericarditis had been due to the friction of a very rigid dilated right auricle against the chest wall. Its surface was irregular and firm to the finger.

The foramen ovale was still patent and would admit a No. 6 catheter.

There was an atheromatous thickening of the superior vena cava as it entered the right auricle.

The history of the case is as follows:

William N—, aged 5, admitted under my care on August 31st, 1892. Always delicate, with a cough and shortness of breath. Three months ago a cart tipped up, and the shaft caught him in the left groin. He cried bitterly, and had two attacks of fainting. Three weeks later a great deal of bruising appeared in the left groin, buttock, loin, scrotum, and penis; a week later his left leg commenced to swell. Since then his abdomen has become distended and his limbs have wasted.

When admitted the following notes were made:—The left leg and the left half of the scrotum are very œdematous. The veins on the penis are prominent, tortuous, and distended, evidently carrying on a large collateral circulation. The veins on the left side of the abdominal wall are dilated and tortuous. Some ascites. Pulse 136. The heart is enlarged, the impulse is outside the nipple line in fifth space. Short apical systolic bruit.

Per rectum the left iliac vein was felt to be large but not thrombosed. Liver and spleen enlarged. Urine normal. Put upon digitalis and nux vomica with a laxative.

September 2nd.—Some dulness at base of right chest; increasing œdema. Next day, as the dyspnœa was increasing and there was more fluid in the chest, 12 oz. were drawn off. The fluid clotted slightly and contained numerous large cells 50 μ across, with a large nucleus, which was in some dividing into two.

9th.—Twelve oz. of fluid drawn off with a Southey's tube from the abdomen. He was put upon Tr. Strophanthi, caffein, and diuretics. He improved for some days.

16th.—The œdema has increased, especially in the left leg, which was acupunctured. A triple sound, suggestive of pericarditis, was audible.

19th.—The right side of the heart has dilated greatly since admission, extending one inch to the right of the sternum; the liver has increased in size. The boy is always very drowsy and his cough occurs in severe paroxysms, often followed by vomiting. Orthopnœa.

In October he passed under the care of Dr. Pye-Smith, to whom I am indebted for permission to publish the case.

October 7th.—Paracentesis of abdomen, 7 oz. drawn off; acupuncture of legs.

15th.—Pericardial rub has greatly decreased during the last month.

27th.—Died gradually from cardiac failure.

The blow on the abdomen from the shaft of the cart, five months before the boy's death, undoubtedly injured the vessels at the brim of the pelvis. It is not possible to determine with certainty whether the sac was originated by yielding of the wall of the aorta or middle sacral artery, or of the vein, with an ultimate communication between them. The sac itself is formed by the pouched wall of the vein, which yielding may have been due to the bruising of the vein. It may also be argued that if the aorta at its termination communicated with the vein, the impact of the blood stream would very quickly injure the wall of the vein and cause it to pouch.

There was a considerable amount of dense fibrous tissue enclosing the vessels and the sac. This may account for the fact that a thin sac lasted so long, and may explain why no thrill was felt *per rectum*.

It is interesting to note that the right iliac vein, which lay behind the sac but did not communicate with it, was unaffected, while the left vein which opened freely into the upper margin of the sac was dilated, and its circulation greatly impeded owing to its sudden embouchement into a large sac of blood at high pressure. The ordinary rotational motion of the blood in the left iliac vein would thus at once become erratic, and owing to the higher arterial pressure in the sac the current would be checked. The high pressure in the sac would cause an abnormally vigorous rush of blood into the vena cava and so help the current in the right vein.

The collateral circulation carried along the penis is an unusual phenomenon.

I am doubtful whether I listened for a bruit in the pelvis, but no note has been made.

These arterio-venous aneurysms may occur as the result of injury or disease in connection with any vessels, but the present case is sufficiently remarkable to be worthy of record.

The cause of the cardiac dilatation is also a point of interest.

There was no valvular disease, and no evidence of any rheumatic disease. I am inclined to think that the shock of the accident produced cardiac dilatation from which the boy never recovered. It is also a question as to what effect is produced on the heart when the pressure in the inferior vena cava is greatly raised. In this case the circulation was short-circuited; the blood must have rushed at an increased velocity and at a considerable pressure along the inferior vena cava. Can this have been the cause of the cardiac dilatation?

The atheromatous condition of the superior vena cava is also evidence of the interference with its current by the high pressure in the inferior vena cava. *January 17th*, 1893.

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20. *Sacculated dilatation of varicose vein.* (*Card specimen.*)

By E. C. Stabb.

The specimen shows an aneurysmal dilatation of right internal saphena vein, removed from female aged 21 (just above knee). When standing the swelling reached the size of a small egg, and there was an impulse on coughing. The vein above the dilatation was normal, but it was slightly varicose below.

The vein of the opposite limb was normal. *May 2nd*, 1893.

III. DISEASES, ETC., OF THE ORGANS OF DIGESTION.

1. *A case of lingual ulcer.*

By Wyatt Wingrave.

The patient, a postman aged 50, complains of a sore tongue of five months' duration. A circular, sharply-defined ulcer, of about the size of a sixpence, is seen on the tip of the tongue; the base had a raspberry-like granular appearance under a thin creamy discharge, and is slightly elevated. It is painless, and firm on palpation. The teeth are carious, but the incisors are wanting in both jaws. It commenced as a small red pimple. There is no specific history, but he has lately coughed and expectorated profusely, whilst his weight has diminished. There are no well-marked signs of phthisis, beyond a few scattered râles and audible expiration at the right apex. No bacilli have been found in scrapings of the ulcer, or in the sputum. The progressive history and the clinical evidence indicate a tuberculous origin.

Note.—Shortly after exhibition the patient met with an accident, and was admitted into Guy's Hospital, where he died. The post-mortem showed well-marked pulmonary phthisis, and sections of the tongue proved the ulcer to be tubercular.

December 20th, 1892.

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2. *Lymphatic nævus of the tongue.*

By Charles A. Morton.

I have brought forward this specimen of lymphatic nævus of the tongue, as I can find but few cases recorded in the 'Transactions' of the Society, and very short and imperfect reference to

the subject in the text-books on surgery. Such growths are often thought to be degenerated blood-nævi.

My case very closely resembles one recorded by Mr. J. Hutchinson, jun., in the 'Transactions' of the Society, 1890, p. 79, and the microscopic section of my growth almost exactly resembles the drawing in his paper.

Clinically, the growth presented as a vesicular wart on the dorsum and edge of the tongue, but not extending under the organ. The papillæ were large, and on many were clear vesicles at the tip. The growth was almost the size of a shilling, a quarter of an inch raised above the surface. It occurred on the tongue of a child eight years of age, and had been first noticed a few months after birth.

Microscopic sections show in the papillæ and submucous tissue numerous large spaces, filled with what appears to be coagulated lymph. These spaces in the papillæ are so near the surface that, in some, the wall is only formed by the corneous layer of the epithelium (unless there is an endothelial lining as well); hence the vesicles seen before removal. The spaces only invade the muscle of the tongue to a small extent in some areas of the sections, and they lie very near together in some of the papillæ, so that only a very thin partition separates them. *May 2nd*, 1893.

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3. "*Adenoma*" *of the soft palate.*

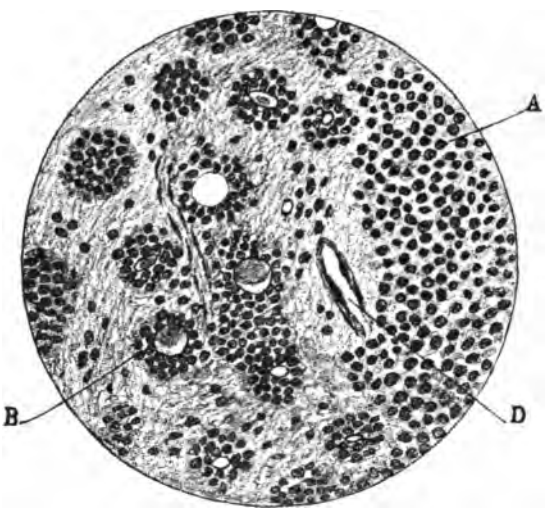
By CHARLES A. MORTON.

THIS tumour was removed by Mr. Harsant from the soft palate of a lady fifty-two years of age. It was noticed accidentally in an examination of the throat. It was distinctly encapsuled, and easily shelled out through an incision made over it. The patient at Mr. Harsant's request also consulted Mr. Stephen Paget.

The enucleated growth was round, the size of a walnut, not lobulated, but slightly irregular on the surface, of a white homogeneous appearance both on the surface and on section, with some patches of redder and more friable granular tissue here and there. It grew, as most palate tumours do, from the left side.

Microscopic examination showed the tumour to consist of somewhat large cells, arranged in some areas in spheres and irregular branching columns, amongst fine, well-formed connective tissue.

FIG. 3.



A. The small-celled growth looking like sarcoma tissue. B. The same forming gland acini. D. A vessel cut across. (⅛th obj.)

In one area there are fairly well formed gland acini, and yet close by, the same kind of cells that form these acini become oval and spindle-shaped like embryonic connective-tissue cells. I do not think we can always judge from the character of individual cells, whether they are epithelial or sarcoma type; for instance, the large round cells of alveolar sarcoma are just like epithelial cells, but I do not know that oval or spindle cells have ever been found to develop from epiblastic cells. Yet in this tumour we seem to have the same cells differentiated into gland epithelium and embryonic connective-tissue cells of oval or spindle shape. Hence we must have an embryonic origin of the tumour from a very early stage in fœtal development. We have, in fact, in this structure of the tumour a strong analogy to that complex nature which is so marked in the ordinary parotid tumour, where we have a growth of tissues both of mesoblastic and epiblastic origin.

And this seems to be the usual feature of these so-called "adenomata" of the soft palate, containing as they do such structures as cell-nests, adenoid tissue, and embryonic connective tissue, as well as gland structure. I have not called this growth an adenoma, because I believe it to be a simple adenoma, but rather classify it with these complex growths—clinically innocent tumours. I regret that it was only noticed accidentally, and so the duration of its growth cannot be stated. That it shelled out so easily is another feature in common with the adenomata, but sarcoma of the palate sometimes shells out easily also. There is one other point in its microscopical structure to which I have not as yet alluded, and that is that in a few areas the well-formed connective tissue is crowded with round or polygonal colourless small cells each possessing a fine black dot. Are these embryonic fat cells? In some parts of the tumour the columns and masses of cells are separated, not by this fine connective tissue, but by an almost hyaline but faintly fibrillated tissue. It is to be noticed that the gland acini have no basement membrane, and that the cells around the gland lumen in some parts are not separated from surrounding areas of cells.

In Sir Wm. MacCormac's case, recorded in the thirty-seventh volume of the 'Transactions' of this Society, Mr. Shattock describes the tumour as consisting of "a hyaline connective tissue, traversed by an irregular branching network of epithelial cells," and in one of Mr. Stephen Paget's cases, recorded in the thirty-eighth volume, an arrangement of branching columns of cells not unlike those in this tumour are figured.

These branching columns are very like the tracks of gland tissue around the little masses of cartilage in parotid tumours. This is more especially so in Mr. Stephen Paget's tumour than in mine.

I think it is not a generally recognised fact that large innocent tumours may develop in the soft palate, and at the same time project externally under the angle of the jaw. Out of the thirty-one cases of adenoma of the palate collected by Mr. Stephen Paget, not one projected externally, though two were so large as to be difficult of removal through the mouth. Mr. Dobson, of Bristol, described in 'St. Thomas's Hosp. Reports,' for 1875, a fibro-adenoma of the soft palate which was so large that it could hardly be removed between the teeth. A lobe of this growth (which occurred in a young woman aged 22), formed a swelling the size of a chest-

nut, under the angle of the jaw. The case has been published with another as "Two cases of Surgical Interest," and has, therefore, been passed over by writers on tumours of the palate. Dr. King, of Hull, had brought a similar case before the Medical Society of London in 1871. In this case "the base of the tumour passed outwards and backwards behind the jaw, where it could be felt externally." This tumour was so large that the mouth had to be slit up to remove it. *November 1st*, 1892.

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4. *Pharyngeal diverticulum.* (*Card specimen.*)

By Samuel G. Shattock.

This is one of the older of the museum preparations; no history is recorded of it; and so inefficiently has it been preserved for microscopical purposes that nothing is discoverable as to the structure of its wall,—whether this, for instance, consists in part of unstriped muscle, or whether there exists any stratum of lymphatic tissue which might indicate its origin from the fore-gut, a stratum such as occurs on certain cervical cysts, and which, in conjunction with a columnar-celled lining, shows their source to be the deeper or enteric moities of the branchial pouches, as distinguished from the superficial or epiblastic invaginations which (except in the case of the hyo-mandibular) do not become continuous with the former. Macroscopically, however, it is an excellent example of a pharyngeal diverticulum which seems to me best explained as having originated in a congenital defect rather than as having been primarily due either to traction from without or pressure from within.

The preparation (No. 900, St. Thomas's Hosp. Mus.) is that of a larynx, with the pharynx and parts of the trachea and œsophagus. Along the left side of the pharynx and œsophagus is a thin pouch, three inches in length, which communicates with the lowest part of the pharynx. The aperture of communication is circular, 1·3 cm. in diameter, and is situate immediately behind the posterior border of the thyroid cartilage, the lower margin of the aperture being ·5 cm. below the level of the crico-arytenoid articulations.

The position of the diverticulum corresponds anatomically with that of the fourth branchial pouch; but whether its origin be congenital or not, the subsequent growth of the sac is unquestionably due to repeated distension; and this holds true in any case, whatever may have been the starting-point of a pharyngeal diverticulum.

Of the specimens in the College of Surgeons Museum, No. 2291 (the well-known one from the bishop) resembles that shown to the Society by Dr. Chavasse (vol. xlii, p. 82), and which is now in the same collection (2291 D). In both, the orifice of the sacculus lies in the *posterior* wall, and at the very lowest part of the pharynx. The histological examination of Dr. Chavasse's specimen showed that it consisted of mucous membrane and vascular connective tissue, with scattered bundles of muscular fibres. An inspection from the right side shows plainly that the protrusion has occurred between the fibres of the inferior constrictor, a portion of which lies above, a portion below, its mouth. Similar protrusions occur in connection with the œsophagus. There are two such in the College of Surgeons' Museum, and a similar one was recorded by Dr. Norman Moore in the Society's 'Transactions' (vol. xxxiii, p. 191). One of the former (2291 B) is described as being probably a traction diverticulum, since its summit is adherent to the trachea.

None of these œsophageal sacculi can be considered as having any morphological significance, and they are paralleled, as are doubtless some of those of the pharynx, by the sacculi met with along the intestine, which sometimes exist in great numbers, as in a case I recorded in the thirty-third volume of the 'Transactions,' page 192; there is another, equally marked, in the Museum of the College of Surgeons (No. 2455 B). A preparation in St. Thomas's Hospital Museum (1028) shows, also, a portion of a colon from which there project numerous small sacculi of the mucous membrane, extruded between the fibres of the muscular coat; many contain pellets of hardened fæces. From a woman, aged 69, who died of cancer of the stomach, liver, peritoneum, and pleuræ; no stricture was detected, nor any other disease whatever of the large intestine.

On the other hand, the congenital forms of pharyngeal diverticulum will correspond, of course, with that of Meckel in the intestine. *May 16th*, 1893.

5. *Epithelioma of the lower end of the pharynx. Growth in bone.* (*Card specimen.*)

By H. D. Rolleston, M.A., M.D.

There was an ulcerated growth extending over the posterior surface of the left half of the cricoid into the wall of the pharynx, as far as the median raphe. The growth did not encroach on the larynx, and did not narrow the lumen of the pharynx to any extent. There was a mass of growth in the glands on the left side of the neck, in continuity with the primary growth.

There were numerous small white nodules of new growth in the skull; there were none in the brain.

On the right side there were secondary growths in the ribs from the second to the eighth inclusive, most of which had so far replaced the bone for a short distance as to allow the rib to be bent to and fro.

There was also a growth in the body of the tenth dorsal vertebra.

Microscopical sections of the growths showed them to be like the primary growth at the lower end of the pharynx, a squamous-celled epithelioma.

From a man aged 62 years, who died in St. George's Hospital, under the care of Mr. Bennett. There were also secondary growths in the liver, aortic glands, and kidney. In one kidney there was a large hæmorrhagic tumour, the cells lining the blood-spaces being large and like renal cells, and filled with fat. An account of this curious tumour will be published later.

November 15th, 1892.

6. *Sarcoma of the œsophagus, with secondary growths in bone.*

By H. D. Rolleston, M.A., M.D.

The œsophagus for its lower three inches is greatly narrowed and thickened by a firm growth which infiltrates the entire circumference. The walls are three quarters of an inch thick. The mucous coat is ulcerated for a great part, but not for the whole, of the extent of the growth. The growth is adherent to the pericardium, but has not penetrated it; it is firmly united to the right lung.

Towards the upper part of the stricture there is a fistulous passage through the growth into a gangrenous cavity in the lower lobe of the right lung. The lower lobe is solid, grey in colour, and œdematous. The glands at the bifurcation of the trachea are swollen and œdematous, one is infiltrated with new growth; there is also an enlarged infiltrated gland on the root of the right lung.

The growth does not extend into the cardiac end of the stomach; attached, however, to the cardiac end of the stomach is an enlarged lymphatic gland, infiltrated apparently with new growth; it was not examined microscopically. The whole naked-eye appearance resembled that ordinarily seen in epithelioma of the œsophagus.

Microscopically the narrowed part of the œsophagus was seen to be infiltrated by a growth composed of medium-sized round cells. Towards the edge the growth was seen to begin in the submucous coat, and to pass underneath the mucosa, and outwards into the muscular coat, which was extensively, but irregularly, infiltrated with nodules of growth. These nodules, so large as to be visible to the naked eye, were composed of uniform round cells, in which there was no alveolar arrangement. Spreading from these nodules, as from centres, the round cells infiltrated the muscular fibres around. When invading the muscular tissue an appearance not unlike that met with in parts of carcinomata was produced. But examination of numerous sections, both of the primary and of the secondary growth, indicate that the growth is a round-celled sarcoma, and not a spheroidal-celled carcinoma.

There was a fluctuating tumour, very vascular on section, in the

left hypochondrium, growing from and replacing the substance of the eighth, ninth, and tenth ribs. This tumour was the size of one's fist. The sixth rib on the same side showed a fracture from the presence of a small mass of growth, which, like the larger tumour, was in the anterior axillary line. The right first rib was extensively infiltrated with new growth, which had grown down into the first intercostal space, and was beginning to attack the second rib. In the right iliac fossa there was a large soft mass of vascular new growth, which had eaten its way through the ilium, and which, in part covered over by the remains of the bone and periosteum, had infiltrated the glutei on the outer side and the ilio-psoas internally. These growths microscopically were seen to resemble that in the œsophagus in the size of the cells, but, in addition, contained much extravasated blood.

There was a vascular soft growth in the right middle fossa of the skull, lying under the dura mater of Meckel's space, and projecting through the temporal bone into the temporal muscle externally. Internally it had not perforated the dura mater.

The brain was healthy.

There were no growths in the spine, the lungs themselves, heart, liver, kidneys, supra-renals, spleen, thyroid, or testes.

There was a mass of enlarged glands, infiltrated with new growth, around the pancreas, but that organ was itself healthy. There was also an enlarged gland near the cæcum.

The patient was a man, aged 54 years, who was admitted into St. George's Hospital under Dr. Ewart's care, to whom I am indebted for permission to bring the case forward. For nine months before his death he had noticed a difficulty in swallowing, and for two months he had been aware of a painless tumour in the left hypochondrium.

Until four days before death the temperature had been non-febrile; it then rose to 102°, and he was two days later found to have the signs of pneumonic consolidation at the right base.

It appears to be almost certain that in this case the sarcomatous growth in the œsophagus was the primary growth, and those in the bones and glands were secondary, both from the history and from the appearance of the respective growths, that in the œsophagus being much the firmer and harder than those elsewhere.

The improbability of a secondary growth occurring in the coats of the œsophagus is great.

Sarcoma of the œsophagus must be very rare; the only recorded cases that I have come across are one by Dr. Chapman, quoted by Mr. Butlin ('Carcinoma and Sarcoma,' p. 160), one shown by Mr. J. H. Targett before this Society ('Path. Trans.,' vol. xl, p. 76), and one by Dr. Shaw, recorded in the same 'Transactions,' vol. xlii, p. 90. In none of these were there secondary growths found in the skeleton. *February 7th*, 1893.

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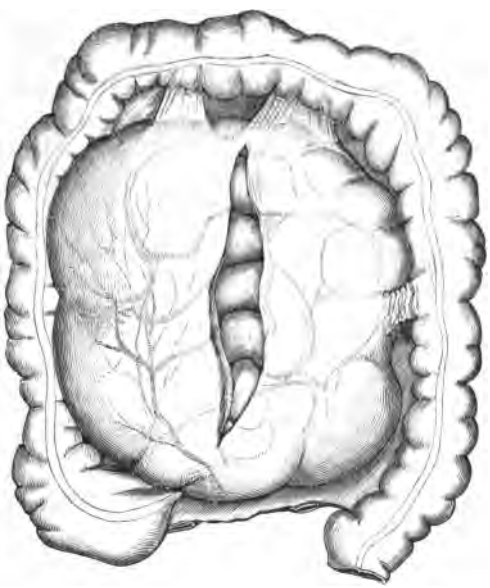
7. *A case of retro-peritoneal hernia.*

By J. Jackson Clarke, M.B.

A man of middle age was admitted under Mr. Page into St. Mary's Hospital with the symptoms of acute intestinal obstruction. He said he had been seized with sudden pain which caused him to fall down in the street.

Mr. Page performed abdominal section. This revealed a large,

Fig. 4.



The hernia seen from the front, with the sac opened to show the contents. The ascending transverse and descending colon surround the sac. Several processes of peritoneum pass from the sac to the colon.

smooth, rounded formation which had the appearance of an ordinary ovarian cystoma, but gave a tympanitic note. After passing a hand into the abdominal cavity, Mr. Page was able to withdraw the whole of the small intestine from an aperture at the lower part of the hernia. The patient gradually sank after the operation.

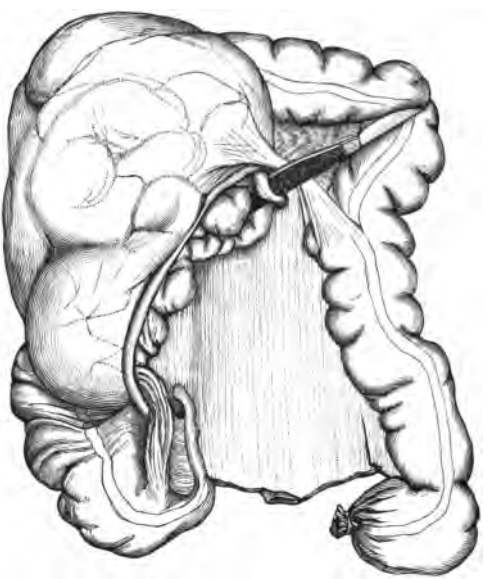
Examination after death revealed a large flaccid sac lying to the left of the back part of the mesentery, and consisting of a double layer of peritoneum. Into this the whole of the small intestine could easily be replaced.

After this had been done the parts had the appearance shown in Fig. 4.

About one foot of the upper part of the ileum was deeply congested; the rest of the small intestine had a normal appearance. The congested portion had probably been twisted within the sac.

By raising the lower and left portion of the hernia upwards and to the right, the arrangement of the parts at the neck of the sac could easily be seen. They are represented in Fig. 5. The aper-

FIG. 5.



The sac has been drawn up and to the right. The ridge which bounds the opening into the sac contains the superior mesenteric artery, which has been freed above, and is shown crossing the handle of a scalpel.

ture easily admitted the closed hand. In front it was bounded by the superior mesenteric artery covered by peritoneum; behind by the peritoneum covering the posterior abdominal wall as it passed to form the posterior portion of the sac, ceasing at the inner edge of the ascending colon. The transverse mesocolon was present, but short, and it was attached in the usual manner. The duodeno-jejunal fossa was absent.

The condition had the appearance of being congenital, and the facts might be expressed by saying that the small intestine had been rotated through 360°, first to the left, then backwards, next to the right behind the superior mesenteric vessels, and lastly forwards. The sac may be described as consisting of that part of the mesentery which lies between the superior mesenteric vessels and the attachment of the mesentery to the posterior abdominal wall.

The whole of the third part of the duodenum was to the right of the superior mesenteric vessels, and possessed a short mesentery.

The case is evidently an example of what Astley Cooper termed the mesenteric variety of retro-peritoneal hernia.

Mr. Page has drawn my attention to a quite similar case published by Dr. Barrs,[1] who refers to a paper by Dr. Pye-Smith ('Guy's Hospital Reports,' vol. xvi, 1870–71, p. 131), where an analysis of cases of retro-peritoneal hernia is to be found.

May 16th, 1893.

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8. *Femoral hernia. Perforation of Gimbernat's ligament by a fish-bone causing inflammation of the omentum in the sac.*

By J. Hutchinson, jun.

John M—, aged 60, was admitted into the London Hospital with an old femoral hernia on the right side, which had been painful and larger than usual for some twenty-four hours, and was the size of a goose's egg. There were no urgent symptoms and no vomiting, but the rupture was irreducible and became increasingly painful, so that an operation was done by Mr. Hutchinson on the day after admission.

[1] 'Lancet,' August 8th, 1891, p. 286.

On opening the sac much thin fluid escaped, and the finger detected, towards the neck of the sac, a sharp foreign body which seemed to project from Gimbernat's ligament. It was extracted with forceps and proved to be a fish bone over an inch long. It had perforated Gimbernat's ligament, through which there was a small pus-discharging aperture. The other contents of the sac (omentum much congested and sloughy, or almost gangrenous where the bone had rested on it) were excised. The sinus in Gimbernat's ligament was scraped and a small drainage-tube inserted in it. It was, of course, obvious that the fish bone must have escaped by perforating the wall of some piece of intestine close to the femoral ring, and that it had been forced through Gimbernat's ligament, but as there was not the slightest sign of general peritonitis, and as it was certain the fish bone had been in the femoral sac for some few days (judging by the condition of the tissues round it), it seemed reasonable to expect that the perforation had closed up and abdominal section was decided against.

The patient did well for a fortnight. The wound suppurated a little but entirely closed, and he was able to get up. At the end of this time he had some diarrhœa which, in all probability, disturbed adhesions and started peritonitis, for from this time he had abdominal pain and subnormal temperature, and died, nearly four weeks after the operation, of suppurative peritonitis. It was not ascertained through what part of the intestine the foreign body had escaped.

Such a complication of a hernia is, I venture to think, almost, if not quite, unique. It is probable that the foreign body, which was very strong and could not easily be bent, had travelled down to the cæcum and, meeting with difficulty in passing the ileo-cæcal valve, had perforated the intestine. But, that it should have been driven through Gimbernat's ligament, and happened to lodge in the sac of a femoral hernia, and then, by irritating the contained omentum caused symptoms resembling those of strangulation, is an extraordinary chapter of accidents. It must be understood that there was no intestine in the sac, and that the direction of the foreign body—its shape and pointing downwards with its blunt end fixed in Gimbernat's ligament—proved that it had been expelled from within the abdomen, and that its entrance into the hernia was purely fortuitous.

That the abdomen was not opened and the perforation found at

the time of operation on the hernia is to be regretted, although, from the fact that no symptoms occurred until a fortnight afterwards, it is probable that the patient would have recovered but for the attack of diarrhœa.

I may here briefly record another case of foreign body within a hernia. I operated for strangulated inguinal hernia (on the right side) on a middle-aged man. A loop of small intestine was present in the sac, but considerable difficulty was found in returning this, although the aperture leading into the abdomen seemed large enough. An obstacle was found in the inguinal canal in the form of the vermiform appendix, which on being drawn down was seen to be inflamed and swollen at its base, with a small gangrenous patch over a sharp substance within the appendix itself. The latter was then carefully excised and its base sutured. On opening it a foreign body, the nature of which could not be exactly ascertained, was found to be impacted. The foreign body was a circular disc, like the very hard scales seen on certain fish, with a sharp spine projecting from its centre. This spine had become fixed in the wall of the appendix, and thus produced the small slough.

February 7th, 1893.

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9. *Fat necrosis and its association with pancreatic lesions.*

By H. D. Rolleston, M.A., M.D.

Fat necrosis in connection with disease of the pancreas has attracted little notice in this country. Dawson Williams in the article "Pancreatitis," in the 'Dictionary of Medicine,' 1890, edited by J. K. Fowler, mentions its occurrence. As far as I know the subject is not described in the text-books,[1] and I do not find any record of it in our 'Transactions.'

Langerhans[2] states that in the subperitoneal fat, especially in the neighbourhood of the pancreas, small areas of fat cells may undergo a peculiar change; the fat becomes crystalline, the fat cells die and set up inflammation around them. The small foci

[1] In a text-book of 'Morbid Histology,' p. 299, 1892, Dr. Boyce says, "In acute pancreatitis hæmorrhages and suppuration may be observed as well as necrosis of the surrounding fatty tissue."

[2] 'Compendium Pathol.,' p. 393; also 'Virchow's Archiv.,' Bd. cxxii, p. 252.

may run together and form prominent opaque whitish masses, which can be picked out on the point of a knife. The liquid part of the fat is got rid of, and the fatty acids may remain or may unite with calcium salts, forming a salt of fatty acids.

Langerhans believes that the primary change is necrosis of the fat cells, and that inflammatory changes set up around the necrosed areas may by becoming confluent produce necrosis of the pancreas, which thus becomes separated and may even be discharged into the duodenum. He denies that pancreatic lesions are a cause of fat necrosis. Fat necrosis, he states, is especially likely to occur in very corpulent subjects.

In an experimental research Langerhans injected the extract of the pancreas of a rabbit, or of a dog, into another of the same species, the injection being made under antiseptic precautions into the subcutaneous tissue. In seven cases no result followed, in one case fat necrosis followed locally, and in three other cases suppuration and fat necrosis were produced at the point of injection, but the author admits that bacterial action could not be eliminated.

From these data Langerhans concludes that the primary change occurs in the fat, which is destroyed by the piolin or steapsin in the pancreatic juice, and that the death of the containing cells is secondary to the destruction of the fat in them.

Fitz, of Boston, in the 'Proceedings of the New York Pathological Society,' 1889, published as the Middleton Goldsmith Lecture an exhaustive account of pancreatitis. He gives the literature of fat necrosis.[1] In suppurative pancreatitis fat necrosis is rare, while in hæmorrhagic and gangrenous pancreatitis it is common. In cases of hæmorrhage into the pancreas he states that fat necrosis has been found, but thinks it probable that in these cases there is pancreatitis as well. At the General Medical Congress at Berlin, 1890, Fitz ('Transactions,' Section 3, p. 146) gives it as his opinion that the pancreatic changes were the cause of the fat necrosis; he had been unable to find any record of a case of inflammatory fat necrosis without associated evidence of alterations of the pancreas indicating or suggesting a pancreatitis. But pancreatitis may occur without fat necrosis,—the more superficial the pancreatitis the more likely was fat necrosis to occur. He regards the fat necrosis as due to inflammation extending from the

[1] *Vide* also 'Transactions of the Association of American Physicians,' vol. v, p. 191.

pancreas to the fat tissue. Both Fitz and Langerhans describe the patches of fat necrosis as being surrounded by a small-cell infiltration. Fitz does not regard fat necrosis as occurring exclusively in fat subjects.

The specimens I show are both from cases that occurred at St. George's Hospital under Dr. Cavafy, by whose kindness I am enabled to bring them forward.

Case 1.—H. M—, aged 50, a lady's nurse. On May 27th, 1892, her patient, who was fat and heavy, fell upon her and caused her severe abdominal pain; on the following day she began to vomit and her bowels were confined, an unusual occurrence with her.

On May 30th she was admitted with vomiting and constipation, and in a collapsed condition. The aspect of the case was one of acute obstruction, so much so that the question of laparotomy was raised. But as the cause was thought to be peritonitis, no operation was performed. In a few days although the vomiting continued the bowels became loose. The patient on admission was fat, but owing to continued sickness lost flesh.

On July 29th a fluctuating tumour was noticed in the right hypochondrium. On August 15th the patient died, worn out with vomiting, which nothing had satisfactorily controlled. The urine on one occasion contained albumen, never sugar.

Post-mortem.—Body well nourished, half an inch of fat on the abdominal walls. Abdomen: numerous opaque white areas in the subperitoneal fat, more frequent near the pancreas; their contents are dry and solid, there is no area of congestion around them. There was no recent peritonitis, but the omentum was adherent to the anterior abdominal wall to the right of the umbilicus by recently organised adhesions. There was an abscess partly in the head of the pancreas, partly around it; the abscess had passed behind the first part of the duodenum and the bile duct, and appeared to have just opened into the transverse colon. The abscess had eroded the head of the pancreas, and the contiguous wall of the second part of the duodenum, but had not perforated it. The mucous membrane of the duodenum was normal. The bile-duct was slit up and appeared healthy; there were two small calculi in the cystic duct. The liver, 56 oz., showed marked fatty infiltration, but neither to the naked eye nor to the microscope were there any appearances like those of the areas of fat necrosis met

with in the subperitoneal fat. Spleen 5 oz., soft. Kidneys, R. = 5 oz.; L. = 6 oz., normal. Supra-renals healthy. Œsophagus, stomach, and intestines normal. Vermiform appendix pervious and free. The pancreas weighed 5 oz. Microscopic sections showed that there was an acute interstitial inflammation spreading in from the walls of the abscess, but that there was no general pancreatitis and no sign of any old hæmorrhage into the pancreas.

Case 2.—Alfred E—, aged 30, of spare habit of body, was admitted to St. George's Hospital on August 19th, 1892. The following history was obtained. A year and a half ago a cart passed over his abdomen, but he apparently was not much injured, as he did not have to lie up. Six months ago he was an in-patient at the Westminster Hospital with sudden abdominal pain, otherwise except for occasional attacks of abdominal pain he had been in perfect health.

After dinner on August 19th he was seized with sudden abdominal pain and severe vomiting; the bowels acted once soon after the onset, but after that no fæces or flatus were passed. He was very collapsed. Laparotomy was decided upon, and the abdomen was opened and small opaque whitish masses were seen in the omentum and on the mesentery.

The patient died thirty-six hours after the operation.

Post-mortem.—There were numerous opaque white areas about the size of a split pea scattered over the peritoneum wherever there was an underlying layer of fat: these areas of fat necrosis were smaller but more numerous than in the first case, they were especially numerous near the pancreas. The pancreas weighed 7 oz.; there was recent hæmorrhage around it apparently spreading from the region of the pancreatico-duodenal arteries, it passed over the entire surface of the organ as far as the spleen; the blood had passed into the substance of the head of the pancreas between the lobules, but for a short distance only. The substance of the organ was firm and to the naked eye normal. The most probable origin for the hæmorrhage seemed to be the rupture of a small aneurysm on the pancreatico-duodenal arteries; these arteries were slit up as was the splenic, but nothing to account for the extravasation was found. There was a small adherent thrombus in the middle of the splenic vein not occluding the lumen, of recent *ante-mortem* origin.

Microscopic examination of the pancreas close to the duodenum

showed quite recent hæmorrhage passing for a short distance between the lobules; there was no evidence of acute interstitial inflammation, and the parenchyma was healthy. There was no sign of old extravasation into the pancreas.

This seemed to be then a case of pancreatic hæmorrhage, and not of hæmorrhagic pancreatitis. The stomach, intestines, and vermiform appendix were normal. The peritoneum showed congestion along the lines where the coils of distended small intestine were not in absolute contact, but there was no lymph or further sign of peritonitis. The kidneys, pancreas, and testes were normal. The common bile duct was slit up and was normal, the liver, 52 oz., was normal. The gall-bladder contained numerous small facetted calculi. The spleen, 5 oz., was healthy. Lungs and heart normal.

Remarks on these two cases.—In both these cases the symptoms were suggestive of some form of internal hernia, a point which Fitz has insisted on. In the first the abscess was presumably traumatic in origin, the two small calculi in the cystic duct not playing any part in its production, as in cases of pancreatic abscess recorded by Dr. N. Moore.[1] The course of the first case was chronic, the second case was remarkably acute. In the first case the areas of fat necrosis were larger and less numerous than in the second, in which the small areas possibly had not had time to coalesce. The cause of the pancreatic hæmorrhage was not made out.

Areas of fat necrosis.—The opaque white areas were found in both cases only in the abdominal cavity, and though the fat of the abdominal walls and of the thighs was examined it was found to be normal. Fitz,[2] however, quotes instances of its occurrence in the subpleural, subpericardial, and subcutaneous fat, as well as in the marrow of bone. Very commonly a similar opaque appearance in the interstitial substance of the pancreas is seen; this is probably due to the post-mortem action of the pancreatic juice on the fat cells contained between the lobules, and is quite different from the present condition. The change, though widely spread, was only found where there was subperitoneal fat, and was absent on the intestines generally, though well marked in the appendices epiploicæ. The areas were roughly round, being larger in Case 1. They never occurred in streaks or lines. They were not raised above the surrounding tissues.

[1] 'Path. Trans.,' vol. xxxiii, p. 186.

[2] 'New York Path. Soc. Proc.,' 1889, p. 63.

The masses when examined fresh melted on heating, and stained in osmic acid like fat. Parts of the mesentery were hardened and cut in celloidin: sections showed masses of cells full of opaque, somewhat granular material, which stood up in marked contrast to the clear normal fat cells of the mesentery. The opaque altered cells were in immediate contact with the normal fat cells, the transition being abrupt. The nuclei of the affected cells were not seen; in some cases the contents of the cells were seen to contain rods which did not stain with the aniline dyes, and which were probably fat crystals.

Around these areas of altered fat cells, which from the alteration of the contained fat and from the absence of their nuclei have been considered to be necrosed, in many cases no change was noted, except the presence of a few fat crystals in the otherwise normal fat cells. In some cases, however, there was some small-cell proliferation, not surrounding the whole circumference of the mass, but occurring on one side, usually in contact with a small vessel. The vessels were full of blood, but extravasation of their contents was almost entirely absent.

Remarks as to the causation of fat necrosis.—The two views quoted above are—(1) that the change is a primary one in the fat of the fat cells, which it is suggested may be set up by the action of the fat-splitting ferment of the pancreatic juice steapsin (Langerhans); and (2) that the change is due to inflammation of the pancreas spreading to the subperitoneal fat. Neither of them is entirely satisfactory (Fitz).

To consider Langerhans' theory first. A primary necrosis of the fat in the fat cells has been suggested by Zenker[1] as being due to their excessive growth, which he says is a danger especially likely to occur in fat persons.

Of this there seems little proof, and it appears improbable that a passive structure like fat should so readily die after showing signs of active growth. The suggestion that the ferment steapsin of the pancreatic juice may be responsible for the fat necrosis seems unlikely in the human subject, because the distribution of the necrosed areas occurs out of the direct reach of the products absorbed from the alimentary tract, as in the great omentum. Fat necrosis occurs in isolated round areas, and not in streaks or lines, as it would if it was caused by a fluid carried by the lymphatics or

[1] Quoted by Fitz, loc. cit.

blood-vessels. In Case 1 the liver showed extensive fatty infiltration, but there was no necrosis of the fat globules as there would be if a ferment absorbed from the digestive tract necessarily produced this change. Again in cases of obstruction of the pancreatic duct by malignant growths or calculi, where the pancreatic secretion might be easily absorbed and carried into the general circulation, fat necrosis is not met with.

Fitz's statistical paper is of the greatest value in showing the association between pancreatitis and disseminated fat necrosis. He regards the pancreatic lesion as primary, and the change in the fat cells as due to the extension of the inflammation[1] to them.

This explanation manifestly does not hold for cases such as those of pancreatic hæmorrhage, where there is not only no peritonitis but no inflammation of the pancreas. Peritonitis, though it may accompany pancreatitis and fat necrosis, by no means always does so. Unless peritonitis is uniformly present it is impossible to explain the widely-disseminated fat necrosis in the subperitoneal fat as due to a direct extension of inflammation from the pancreas to the subperitoneal fat.

It might further be urged if fat necrosis be produced by inflammation, why does it not occur in cases of general peritonitis or in the neighbourhood of local peritoneal abscesses? Why, when it occurs, is it associated with suppuration or hæmorrhage about one abdominal organ only, the pancreas?

It is suggestive that the cases in which fat necrosis is found, viz. cases of pancreatic inflammation and hæmorrhage, are clinically characterised by their resemblance to acute intestinal obstruction. The symptoms, vomiting, constipation, and collapse, are the result of some severe shock to the sympathetic. Hæmorrhage around or inflammation of the pancreas is very likely to spread to, or to irritate, the solar plexus, and so give rise to severe symptoms.

It appears not improbable that the changes in the fat cells called "fat necrosis" may be trophic in origin, and due to the disturbance of the abdominal sympathetic which is shown clinically by symptoms like those of intestinal obstruction and by collapse, and that in this way fat necrosis is secondary to pancreatic lesions. The distribution of the fat necrosis is in the area of the abdominal sympathetic. The

[1] 'Trans. General Medical Congress, Berlin, 1890,' "Section of Pathology," p. 146; 'Middleton-Goldsmith Lecture,' p. 64.

limitation of the fat necrosis to the abdominal cavity in at any rate the very great majority of cases is thus rendered intelligible.[1]

October 18*th*, 1892.

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10. *Fat necrosis in a case of fatal peritonitis due to perforation of the gall-bladder.*

By HERBERT P. HAWKINS, M.B.

THIS specimen was obtained from the body of a man, aged 55, who was admitted into St. Thomas's Hospital under Dr. Sharkey, on November 9th, 1892. The patient had always enjoyed good health until November 6th; on this day he was suddenly seized with severe pain in the upper part of the abdomen and soon began to vomit. The vomiting and pain continued with great severity up to the time of admission, and in this time he passed neither fæcal matter nor flatus.

The abdomen was explored on the day of admission by Mr. Clutton. There was found to be acute general peritonitis, and more than two pints of bile-stained fluid were removed from the abdominal cavity. The character of this fluid led to an examination of the gall-bladder, but no perforation in it was discovered. On the contrary it was found to contain bile, and its fundus could be made tense by pressure. The patient died thirty-six hours later.

At the *post-mortem* examination there was abundant evidence of recent acute peritonitis, and this was found to have been due to a leakage of bile from the gall-bladder. The gall-bladder contained bile but it was not distended; on running the bile towards the base of the bladder with the finger, the fundus could be tensely distended without any apparent leakage. On opening it, however, there was found an ulcer situated to the right of the fundus, oval in shape,

[1] Since this paper was read I have met with two cases in which there were conditions of the pancreas which might have been associated with fat necrosis, but in which that condition had not occurred. In one there was extensive recent hæmorrhage around the pancreas in a man aged forty-one years, who had had jaundice for a considerable time as the result of columnar-celled epithelioma of the common bile-duct. The other case was one of multiple suppurating foci in the pancreas, secondary to impacted calculi in the common bile-duct (*July* 19).

one third of an inch in its long diameter; and from the floor of this a small passage ran obliquely to open on the surface by a minute orifice. The neck of the gall-bladder contained an impacted oval gall-stone half an inch long.

The omentum, which was somewhat thickened and rolled up, the mesentery, and to a less extent the parietal peritoneum, showed numerous spots, nodules and small masses of a bright yellow material, which from their distribution and arrangement were obviously altered fat lobules. In the omentum could be seen normal fat lobules side by side with yellow lobules of precisely similar shape and arrangement; and in some places small points of this commencing change could be seen in lobules which were otherwise normal. The progress of the change could be well seen in the fat of the mesentery of the vermiform appendix. The largest masses occurred on the ascending colon, where they represented appendices epiploicæ which had *in toto* undergone this change. There were none on the descending colon or sigmoid flexure. In the omentum there were several cylindrical yellow masses half an inch long, interspersed among numbers of small nodules and points, which had the size and shape of the granules of fat usually met with in this position. These were mostly aggregated in a patch of two or three square inches lying to the right of the middle line. Groups of similar nodules up to the size of a split pea occurred in the mesentery, being especially numerous at the intestinal border, but in no instance occurring on the intestine itself. There were a few patches on the parietal peritoneum, some of which were a quarter of an inch in diameter. There was a large thick patch as well as several small nodules on the peritoneal surface of the gall-bladder, but these had no relation to the ulcer therein. There were a few yellow points in the fat around the pancreas, but no change in the renal fat. There was no change in any fat but that which was underlying the inflamed peritoneum.

The colour of this material was not the dead white described in similar cases, but a bright yellow, being stained by the bile which was free in the abdominal cavity. The material had a rather greater bulk than the fat which it replaced, so that the patches on the parietal peritoneum were distinctly raised above the surface. It was somewhat firmer than caseous material, and on incision into a nodule it could scarcely be squeezed out.

The body was that of a well-built and well-nourished man, and it

contained a moderate amount of fat. There was no other disease in the body and neither pancreas nor liver showed anything abnormal, save slight fatty change in the latter.

Under the microscope the nodules were seen to consist of crystallised fat, the original capsule being filled with indistinct feathery tufts. Immediately around each nodule was a zone of distended capillaries and a definite round-celled infiltration, which formed a complete boundary between it and the adjoining healthy fat. This inflammatory reaction around it may be taken as showing that the morbid change in the fat was of recent occurrence, and that it had acted as a foreign body.

This change was first described by Bulser[1] under the name of fat necrosis, and it is fully considered in the monograph of Fitz[2] on the subject of acute pancreatic disease. On October 18th, 1892, Dr. Rolleston contributed a paper on the subject to this Society. He described two cases, in both of which there was disease of the pancreas, in one case an abscess in its head, in the other hæmorrhage around it; and in respect of this association with some affection of the pancreas his cases resemble those described by Fitz and by Langerhans.

Neither cause nor effect of this sudden morbid cystallisation of subperitoneal fat is certainly known. Its occurrence in connection with acute disease of the pancreas is far too frequent and uniform to be passed over as a mere coincidence. But it certainly occurs when the pancreas is perfectly healthy, as in the specimen shown, and most commonly in cases of peritonitis. Here it would seem to be a local change secondarily induced by the inflammation of the overlying peritoneum, and I have recently examined a case of perforation of the rectum and localised pelvic peritonitis, where fat necrosis was well marked, but was absolutely limited to that part of the peritoneum which was inflamed.

At any rate, notwithstanding the rarity of the condition, it has a certain clinical importance. For in the necessarily imperfect examination which is allowed by exploratory abdominal section, it would be by no means difficult to mistake these raised nodules and patches for tubercular or malignant disease.

November 15th, 1892.

[1] 'Virch. Archiv,' 1882, p. 520.

[2] 'Middleton-Goldsmith Lecture,' 1889.

11. *Fat necrosis of peritoneum.* (*Card specimen.*)

By CECIL F. BEADLES.

THE subject of "fat necrosis" has already twice been brought before the Society during the present session, viz. by Dr. Rolleston and Dr. Hawkins, the former entering fully into the literature of the subject. As, however, there appear to exist many points of doubt as to the pathology of this condition, it may not be without interest to add the following brief note of a case which has since come under my observation.

A female lunatic, aged 53, whose insanity was only of about two months' duration, died after fifteen days residence in Colney Hatch Asylum. She was said to have delusions of royalty and of a grandiose nature, was very talkative, incoherent and restless. She also stated that she was going to die. On admission into the asylum her health was much impaired, and she had cardiac symptoms. She remained confined to bed. On the 15th day she had profuse hæmatemesis which left her in a very exhausted state, and she died the following day.

Post-mortem.—The stomach was the seat of a large chronic ulcer on its posterior wall, and contained much clotted blood. In the perisplenic fat was a large mass of what appeared to the naked eye to be a deposit of caseous substance, somewhat resembling tubercle undergoing degeneration. Numerous small whitish deposits of a similar character studded the omentum. The spleen was somewhat enlarged. Beyond the fact that the heart was rather soft and fatty, the liver fatty, and the brain substance somewhat tough, there was nothing further to note. The arteries were healthy. No abnormality was noticed about the pancreas. The body was fairly nourished.

Microscopically, the small areas in the omentum and the large mass near the spleen were seen to be due to necrosis of the fat, a minute description of which Dr. Rolleston has already given. This is the only case which I have seen, and I do not believe it has before been described in lunatics. In this case there does not appear to be any history of an injury. *May 2nd*, 1890.

12. *Sarcoma of the peritoneum, invading the colon from without, and causing fatal perforation.* (*Card specimen.*)

By CHARLES A. MORTON.

PATIENT, aged 73, was on November 17th, 1892, attacked with abdominal pain and vomiting. He was not seen by his medical attendant, Dr. Lees, until the 20th, when he was dying. During the 20th he vomited large quantities of brownish fluid. There had been no action of the bowels until after an enema, just before he died. A year before Dr. Lees had attended him for abdominal pain and vomiting, for a short time. At this time it was doubtful whether there was a swelling in the right side of abdomen. In the interval between the attacks he remained fairly well.

Post-mortem.—I made the post-mortem examination twelve hours after death. The abdomen was enormously distended with tympanites, and on making a small opening into the peritoneum, a large quantity of gas escaped of extremely foul smell. On laying open the cavity, the omentum was seen to be blackened all over, and on its surface were several minute nodules. Connected with its left side was a large broken-down mass of tumour tissue and blood-clot, which felt like a very soft placenta, but more broken-down. Loosely connected with it were several round smooth growths of all sizes, from a pea to a duck's egg, very dark on the surface, but white inside though speckled with black patches, and so soft that they broke upon any but the gentlest handling. They were all of the same character. One was as large as a cricket ball, quite round and smooth, with the same slate-black exterior, but within it contained a broken-down mass of tissue and clot. The mass of broken-down growth invaded the wall of the descending colon in its upper part, and a portion of the growth, about the size of a marble, penetrated into the bowel, pushing before it the mucous membrane, just like the finger pushing a cloth in front of it. Close to this penetrating growth, in the wall of the bowel was a perforation the size of a threepenny piece, leading into the broken-down tumour tissue, and hence into the

peritoneal cavity. The edges of the opening were like those of an ulcer, round and cleanly cut. The gut was not narrowed at this part. Above, it contained a little quite liquid fæcal matter; below, none, though there were a few small masses of solid fæcal matter in the rectum.

The intestines and parietal peritoneum were stained a light brown colour; and there was a pint of black thick fluid in the pelvis. The omentum was adherent to the parietal peritoneum on the right side of the abdomen, but there was no recent peritonitis, and the intestines were not adherent. Very numerous small, white, firm nodules of growth studded the mesentery, and in one instance grew just on the intestine at the margin of the mesentery, but not on any other part of the intestine or parietal peritoneum.

The liver was not enlarged, but contained a few small, round, very firm, white growths under the surface, but not in the interior. The largest was very markedly umbilicated, but hard throughout. There were no growths in the lungs or other organs. The kidneys were cirrhotic, and there was a fibro-calcareous patch of old tubercle at the apex of one lung.

Microscopic sections of the growths in the peritoneum and liver show the tissue to be spindle- and round-celled sarcoma.

May 2nd, 1893.

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13. *Simple gastric ulcer of unusual form.* (*Card specimen.*)

By Lee Dickinson, M.D.

The stomach of a woman, aged 32, who died in St. George's Hospital under the care of Dr. Dickinson in February, 1893.

The patient had suffered from dyspepsia for eight years, and had been wasting for two years. Towards the end she had frequent slight hæmatemesis. The cause of death was inanition.

The specimen shows a simple ulcer, of annular form, affecting the whole circumference of the stomach in the pyloric region, and encroaching upon the anterior part of the pylorus.

The pylorus is thickened but not stenosed, and its edges project into the cavity of the stomach.

The peculiarities of this ulcer are its annular form, its great extent (best seen when the specimen was fresh), and its probable long duration.

Microscopic examination of the thickened part of the stomach revealed nothing but chronic inflammatory changes.

March 21*st*, 1893.

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14. *Perforating ulcer of duodenum.* (*Card specimen.*)

By E. C. Perry, M.D.

A pylorus with the adjacent parts of the stomach and duodenum laid open to show a perforating ulcer of the intestine. The perforation is oval, measuring half an inch in its longer diameter, and is situated on the anterior wall half an inch from the pylorus. Its edges are abrupt, and the intestinal wall around it is somewhat thickened. Immediately opposite to it on the posterior wall there is a second ulcer, similar in shape and size, the base of which is formed by the thickened serous coat of the bowel.

Frederick G—, aged 21, was admitted into Guy's Hospital under Dr. Perry eighteen hours after the onset of an acute attack of abdominal pain, shortly followed by vomiting and collapse. Laparotomy was performed, and a large quantity of dark brown acid fluid without fæcal odour was evacuated from the peritoneal cavity. There was much lymph upon the coils of the intestine. Four hours later the patient died. *October* 18*th*, 1892.

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15. *Papilloma of the duodenum.* (*Card specimen.*)

By E. C. Perry, M.D.

A portion of a duodenum mounted to show a cauliflower-like mass of growth having a broad attachment to the mucous membrane around and below the papilla of the common bile-duct,

in which a yellow rod has been inserted. The mass is about 1½ inches long, ¾ of an inch broad, and projects ½ inch above the surrounding mucous membrane. Close to the growth at its upper part there is a small shallow pouch in the wall of the duodenum.

Elizabeth D—, aged 49, was admitted into Guy's Hospital under Dr. Goodhart for jaundice with pyrexia, and hæmorrhage from the intestine. She died three months from the commencement of her illness. At the autopsy two chronic ulcers were found in the rectum, and there were numerous small abscesses in the liver. The common bile-duct was not obstructed.

December 6th, 1892.

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16. *Case of multiple papillomata of colon and rectum.*

By Norman Dalton, M.D.

The case was that of a woman, aged 28, under the care of Professor Wm. Rose in King's College Hospital. Eighteen months before admission she had had a polypus removed from the rectum in the Middlesex Hospital, and for six months before admission she had suffered from pain in the rectum with the passage of blood and mucus. The rectum was examined under an anæsthetic and a large mass of soft elongated polypi was discovered. The polypi were removed as far as possible, but the patient continued to bleed from other growths higher up and died shortly afterwards.

At the *post-mortem* there was congestion of the pelvic peritoneum and connective tissue, but no peritonitis. All the organs (including the stomach and small intestine), except the large intestine and rectum, were *normal*. Notably there was no enlargement of glands. The large intestine contained a good deal of blood and was deeply congested. In the rectum there was a ragged area of considerable size from which the tumours had been removed during life, but a few tumours still remained. Each tumour consisted of a peduncle, which in most cases had a bulbous extremity. The peduncles

varied in length from 2½ inches downwards, some being round and others flattened. The bulb at the end of the peduncle was soft and, in many cases, bleeding. The base of the peduncle was not thickened and the mucous membrane moved freely over the muscular coat of the bowel. Further, on cutting into this base nothing like a tumour could be seen infiltrating the connective tissue. In the sigmoid and colon isolated growths were found here and there, but a considerable group of them was found in the upper part of the cæcum.

Microscopical examination confirmed the idea that the tumours were simple outgrowths, for the mucous membrane passed smoothly from the peduncle to the level of the bowel, and there was no growth in the submucous connective tissue. The stalk consisted of fibrous tissue and large blood-vessels, and was covered by the mucous membrane, which did not here show any adenomatous proliferation. The bulbous extremity was also made up of connective tissue and blood-vessels with some free blood; but here there was a slight amount of proliferation of gland tissue. The growths, therefore, were partly papillomata and partly adenomata; the former predominating.

The mucous membrane between the growths was in a state of slight catarrh, and the solitary glands showed simple enlargement.

Thanks to Mr. Leopold Hudson, of the Middlesex Hospital, I am able to add that in December, 1890, there was only one growth in the rectum, and that its structure was practically the same as those which I have examined.

Remarks.—These cases are not very common, but several have now been placed on record in the 'Transactions' of this Society. In some of the recorded cases the polypi have been principally adenomata, in others papillomata. The distinction is important from a histological point of view; but, practically they are all outgrowths of the mucous membrane and submucous tissue, and contain in varying proportions the elements of those tissues. That is, there is increased growth of connective tissue, and blood-vessels, and of glands, sometimes more of the one and sometimes more of the other. In the analogous villous tumour of the bladder the glandular element is practically omitted because the bladder mucous membrane is not so rich in glands as that of the intestine.

Although these polypi return so frequently after removal, they

are not, strictly speaking, malignant. Still Dr. Handford and Mr. Harrison Cripps have brought forward cases to show that they may become so, *i. e.* instead of merely growing out from the mucous membrane as is their natural tendency, they have grown inwards, infiltrating the muscular coat and reproducing themselves in other organs. Histologically all cancer may be regarded as badly-formed gland tissue; so that, although we do not know what occasions the change, still it is not surprising that a tumour with gland elements in it should take on the characters of a cancer. A good instance of this change from innocence to malignancy on the part of a tumour occurs in the ovary; for in many cases of cancer of the ovary and peritoneum, the structure of the growth in the ovary is identical with that of the true ovarian cystoma, which is not, in the vast majority of cases, malignant.

The origin of the disseminated polypi is, of course, obscure. They are quite as likely to be caused by parasites as any other tumour. I have not been able to find, in all cases, that they were preceded by catarrh of the bowel, so that does not seem to be a predisposing cause. They are not congenital, as they have been known to develop after others have been removed; but it is probable that a congenital predisposition to their growth exists, as Mr. Harrison Cripps has reported three cases which occurred in one family. Lastly, it has struck me as curious that these polypi which so much resemble enormously hypertrophied villi; should occur in such numbers only on surfaces where there are naturally no villi, *i. e.* on the mucous membrane of the large intestine and rectum, the bladder, and the uterus. I would, therefore, throw it out as a suggestion that one factor in their growth may be some developmental mistake, which, from some cause or other, becomes conspicuous in after life. *January 17th*, 1893.

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17. *Colloid carcinoma of the stomach.* (*Card specimen.*)

By E. C. Perry, M.D.

A stomach laid open to show a slightly raised ulcerated growth, extending from just within the pyloric ring along the lesser curvature for a distance of five inches, and affecting both walls of

the organ, so as almost to reach the greater curvature. The edges of the growth are raised and slightly everted, and its ulcerated surface presents numerous gelatinous-looking nodules. On the reverse of the specimen the growth is seen to project as a lobulated mass beneath the serous coat. Histologically it is a spheroidal-celled carcinoma, many of the cells of which have undergone colloid degeneration.

Elizabeth P—, aged 81, was admitted into Guy's Hospital, under Mr. Davies-Colley, for a strangulated femoral hernia, from which she died. There was no history of vomiting or other symptom which might be referred to disease of the stomach until the onset of the intestinal obstruction, four days before her death. At the autopsy no secondary deposits were found.

No case of malignant disease of the stomach in the female has hitherto been reported in the 'Transactions' of the Society in which the patient was more than 72 years of age.

November 15*th*, 1892.

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18. *Malignant disease of the upper part of the jejunum ulcerating into the ascending colon.*

By Arthur Voelcker, M.D.

From a man aged 33 years, who was admitted into Middlesex Hospital under the care of Dr. Coupland.

The upper part of the jejunum is the seat of a soft ulcerating new growth, which is situated in the submucous layer. The gut itself at the seat of new growth is much dilated.

The ascending colon is adherent to the growth, which has ulcerated into it.

No secondary deposits were found in the internal viscera.

The man seemed quite well till October 21st, when he had an attack of diarrhœa, which continued until his death on November 12th.

November 15*th*, 1892.

19. *Secondary sarcoma of the ileum.* (*Card specimen.*)

By E. C. Perry, M.D.

THREE portions of an ileum, each of which shows a discoid mass of growth projecting about a quarter of an inch above the mucous membrane. The largest measures two and a half inches, and the smallest about one inch in diameter. In all the specimens the growth presents an ulcerated surface, and in one it has penetrated the wall of the intestine, which has yielded, producing a globular pouch projecting externally. In the wall of the pouch there is a perforation, which in the recent state was closed by adhesion to a neighbouring coil of intestine. Histologically the growth is a round- and oval-celled sarcoma.

William L—, aged 45, was admitted into Guy's Hospital, under Mr. Jacobson, for a growth in the right tonsil, which was first noticed seven weeks before admission. He died about six weeks later, and at the autopsy secondary deposits were found in the cervical and bronchial glands and in the pleura.

November 15th, 1892.

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20. *Columnar epithelioma of the small intestine, in a young woman aged 23.* (*Card specimen.*)

By Charles A. Morton.

E. L—, aged 23, was in the Bristol Children's and Women's Hospital, under the care of Mr. Dacre, suffering from chronic intestinal obstruction. A fairly defined lump the size of a walnut could at times be felt in the left iliac fossa. On September 29th, 1892, an incision was made there, and some distended small intestine traced down to a thickened adherent mass in the iliac fossa, which felt like a new growth. It was too adherent to remove, and so the small intestine was simply stitched to the wound, and

subsequently opened. The obstruction was completely relieved, and she went home on November 4th, but the swelling in the iliac fossa increased in size. A few days after leaving the hospital the symptoms of obstruction returned, and she died on December 25th.

A *post-mortem* examination was made by Mr. Freeman, the medical man who attended her after she left the hospital, who kindly forwarded to me the portion of adherent bowel from the left iliac fossa. In the lower end of the ilium is a marked constriction around the gut, as if a piece of string had been tied around it, and the wall of the gut, for half its circumference, is thickened by new growth. The lumen of the gut is very much reduced in size, and the interior is ulcerated over the area of new growth in its wall. Microscopic sections of the whole thickness of the wall, where infiltrated by the growth, show alveoli, lined with columnar cells.

May 2nd, 1893.

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21. *Meckel's diverticulum perforated by numerous ulcers.* (*Card specimen.*)

By G. H. Makins.

The diverticulum was removed by operation in a case (fatal) of acute intestinal obstruction, of six days' standing.

The removed portion is extinguisher-shaped in outline, two inches long, and about one inch in diameter at the end which joined the bowel.

The remarkable feature consists in the presence of thirteen perforations, which convert the diverticulum into a kind of sieve. Some of the openings are sharply cut, others are bevelled at the expense of the mucous coat, suggesting possible perforation from within.

These perforations might either be the result of a process of ulceration resembling that occurring in perforating appendicities; if this were the case, the swelling of the diverticulum might have been the direct cause of the strangulation.

Or the perforations may have been due to local gangrene, resulting from the tension to which the diverticulum was subjected.

January 17th, 1893.

22. *Cirrhosis of the liver, pancreas, and kidney, from obstructive valvular disease of the heart.*

By Percy Kidd, M.D.

Henry S—, aged 16, weighing machine attendant, was admitted into the Brompton Hospital, in June, 1885, with the following history. At three years of age he had fits, and when seven years old he had chicken pox and chorea, followed by rheumatic pains.

In November, 1884, he had to leave his place, owing to weakness and "pain at the heart." On admission there were signs of enlargement of the heart, with double mitral and double aortic murmurs, and the liver was enlarged. The patient left the hospital after two months.

In February, 1887, he was readmitted, on account of dropsy and dyspnœa, which had developed after a "bilious attack" three weeks previously.

The patient was now very dyspnœic, the liver was considerably enlarged, and ascites was present, together with œdema of the lower extremities. The heart was greatly enlarged, and a well-marked pre-systolic thrill and murmur were noted at the apex, as well as a systolic murmur at the base. Paracentesis abdominis gave some relief. Ecchymosis and a bullous, discharging eruption appeared on the right thigh, and subsequently ulcerated. The patient gradually sank from exhaustion seven weeks after admission.

Autopsy.—Body extremely wasted and very small, not larger than that of a boy of ten or twelve years of age. Ascites. Hydrothorax on right side.

Heart.—Weight, 24 oz. Pericardium adherent. Great hypertrophy and dilatation of both auricles. Marked hypertrophy of left ventricle, and to a less extent of right ventricle. Aortic and pulmonary valves competent to water test. Both arterial orifices enormously contracted, the valves greatly thickened and adherent. The aortic cusps calcified. No vegetations. The pulmonary orifice admits a large cedar pencil, the aortic opening still smaller.

Mitral and tricuspid orifices of buttonhole shape, the valves greatly thickened and contracted. Two soft vegetations on the auricular aspect of the mitral.

The mitral orifice admits a large probe, the tricuspid admits the forefinger.

Muscle of the heart healthy.

Foramen ovale closed. No trace of ductus arteriosus.

Liver (weight, 4 lbs.) large, tough, and congested. Surface smooth. On section hepatic veins widely gaping and thickened. Gall-bladder thick and contracted.

No calculi here or in ducts.

Pancreas rather large and extremely hard.

Spleen (weight, 3 oz.) small and tough.

Kidneys (weight, 10 oz.) much indurated and of deep red colour. A depressed yellow infarct in one kidney.

Microscopical examination.

Liver: In all sections the capillaries are much distended, separating columns of more or less atrophied liver cells. In many places the capillary walls show hyaline thickening. Where the disease is more advanced individual liver cells are separated by a thick hyaline trellis-work of connective tissue, and numerous "pseudoducts," or string-like collections of short cubical granular cells, are seen embedded in a granular hyaline framework. In a few places a hyaline fibroid thickening of the interlobular tissue, marked with a varying number of nuclei, can be detected; but in no part is there any distinct fibrillation.

Pancreas: The lobules are marked out irregularly by a thick hyaline connective-tissue growth, and a similar thickening of the membrana propria of the individual gland tubules is seen in many parts. In some places the pancreatic cells have almost completely disappeared, leaving a coarse hyaline meshwork, the angles of which are occupied by enormously-distended capillary vessels.

Kidney: Sections show a slight diffuse intertubular connective-tissue thickening like that seen in the liver and pancreas, the cirrhotic change being most pronounced in the peripheral portions of the cortex.

The interest of the case centres in the ætiology of the cirrhotic process. Inquiries were made as to the possibility of alcoholic excess or syphilis, but no evidence pointing in this direction was forthcoming. It is, of course, freely admitted that the exclusion

of these two factors is almost impossible, but this method of causation is rendered improbable by the fact that the morbid change involves not only the liver, but also the pancreas and kidney.

The conclusion is therefore suggested that the fibrous change in these organs is the result of vascular obstruction. The degree of obstruction here present, involving all four valvular orifices of the heart, is, in the writer's experience, quite unique, and the highly-undeveloped condition of the patient's body amply testified to the extreme and long-standing derangement of the circulation.

October 20th, 1892.

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23. *Cirrhosed liver from a youth aged sixteen.* (*Card specimen.*)

By F. Penrose, M.D.

The specimen was obtained from the body of a boy, aged 16, who died quite suddenly, November 24th or 25th, 1892.

The only history obtainable was that he was the son of extremely poor parents, who, according to the statement of the coroner's officer, were not in the least likely to have given him any drink; and that he was known to have had an enlarged liver in early childhood, "as a doctor had said so."

The *post-mortem* examination, made on November 28th, the weather being very cold at the time, showed that death was due to hæmorrhage from the portal venous system, especially from the veins of the omentum.

The liver was hobnailed, cirrhotic, and enlarged. There were no abnormal adhesions of the liver due to peri-hepatitis. The spleen was enlarged, but otherwise apparently healthy.

No traces of syphilis could be found in any other part of the body, though carefully looked for, and all the other organs of the body appeared healthy. The body was well nourished.

December 6th, 1892.

Report of the Morbid Growths Committee on Dr. Penrose's specimen of cirrhosed liver from a youth aged sixteen.—The liver is very irregular on the surface, presenting both small and large nodules, and much thickening of the capsule in the depressions between them. There are no gummata. On section the capsule seems to be universally thickened, and from it run broad strands of fibrous tissue into the substance of the organ. Near the surface there are large areas where the liver tissue is entirely obliterated by fibrous tissue, and a little deeper islets of liver cells can be seen closely surrounded by it. Further still from the capsule the liver (except that it is decomposed) appears to be healthy.

The striking point seen in microscopic preparations is the great extent occupied by well-developed masses of connective tissue which, near the capsule, contain no relics of the liver structures except bile ducts. Around the deeper lobules which are still visible the connective tissue is in marked excess, but less so than near the surface.

Sections of the deeper parts, which appeared to the naked eye to be simply decomposed, present strands of thick fibrous tissue surrounding groups of lobules. The disease is evidently cirrhosis of the ordinary multilobular variety, reaching its maximum in the neighbourhood of the capsule; but there is nothing in the history of the case, or in the characters of the liver itself, to show in what way the disease arose.

SEYMOUR J. SHARKEY.
F. S. EVE.

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24. *Ulceration of biliary calculi from the gall-bladder into the duodenum and transverse colon.* (*Card specimen.*)

By ARTHUR VOELCKER, M.D.

FROM a woman, aged 42, admitted into Middlesex Hospital, who died with carcinoma of the breast, and who had no history of any biliary colic or jaundice.

The gall-bladder contained yellow bile, but no calculi.

Near the fundus is a depressed scar. The transverse colon is

adherent to the gall-bladder at this situation, but there is no opening now between the two.

At the neck of the gall-bladder is another small scar with a small fistula, through which a glass rod is passed leading into the duodenum.

The cystic and bile ducts are natural. No ulceration about the papilla.

The liver is slightly fatty. *March 29th*, 1893.

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25. ***Primary carcinoma of the head of the pancreas; thrombosis of the left iliac vein and pampiniform plexus; pulmonary embolism.*** (*Card specimen.*)

By Arthur Voelcker, M.D.

From a woman, aged 63, who was admitted into Middlesex Hospital on October 27th, 1892, under the care of Dr. Douglas Powell.

The left leg had suddenly become swollen a few days previously. The patient had got out of bed and was found kneeling at the bedside. She was pulseless, and died in a few minutes. She had been subject to "bilious attacks" for two or three years, but had only felt really ill since October 1st, 1892.

There is a hard pale mass of carcinoma in the head of the pancreas, and numerous secondary nodules in the liver and neighbouring lymphatic glands. There is no new growth in the stomach. There are seven calculi in the gall-bladder, but no obstruction in the hepatic, cystic, or common duct.

There is a partially adherent decolourised clot in the left iliac vein, and the left pampiniform plexus is thrombosed.

There are two small infarcts in the lower lobe of the right lung posteriorly, and one larger one in the left lower lobe.

A large thrombus is seen in the left pulmonary artery.

November 1st, 1892.

IV. DISEASES, ETC., OF THE GENITO-URINARY ORGANS.

1. *Sarcoma of the kidney with loose masses in the bladder.* (*Card specimen.*)

By F. G. Penrose, M.D.

The following case appears to be worthy of placing on record for the reasons: (1) that accurate information as to the character of the kidney tumour was given by the urine; (2) that the cast mentioned below bore such a striking resemblance to a worm.

R. W—, aged 52, was admitted into St. George's Hospital under the care of Mr. Bennett, on February 7th, 1888. Mr. W. C. Bull, who was then Surgical Registrar, obtained the following history: The patient said that he had been in perfect health until June 28th, 1886, when he woke up to find that he was nearly blind.

Three weeks before Christmas, 1887, he began to have difficulty in getting urine to pass. The stream would stop suddenly, and only after some time the water began to dribble away. On January 23rd, 1888, after having strained hard to pass water, and having been in great agony, he found a long worm-like body issuing from the meatus, and in about a quarter of an hour this mass was shot out of the meatus. Since that time the urine had passed more freely until within a few days of his admission into the hospital, when he had experienced similar symptoms. The mass mentioned above, which had escaped from the urethra, was six inches in length and pointed at both ends, apparently a cast of the urethra. It was submitted to Dr. Delépine, who reported as follows:—"This cast is partly composed of coagulated blood and partly of tumour-cells. The tumour-cells form masses distributed all through the cast. From their nature the tumour is evidently much degenerated,

and as it is difficult to know whether the stroma between them is not partly due to the presence of coagulated blood, it is difficult to say whether the tumour is carcinomatous or sarcomatous in nature (it is probably sarcomatous)."

On admission the patient stated that he had never passed any blood in his urine; that during the last two years he had lost about 3½ stones in weight. He complained of the difficulty with his water, and of a pain behind his left nipple.

In the left half of the abdomen a large irregular tumour was found. It occupied almost the whole of the left loin; it reached as far as the middle line, but did not extend beyond it to the right; it presented all the characters of a renal tumour.

He was found to have advanced optic atrophy; there were several sebaceous cysts of the scalp which had existed for the last twenty years. The urine which was passed on the first day after admission was clear, acid, of sp. gr. 1012, and contained a trace only of albumen. On February 14th the urine was reported by Dr. Delépine to be "acid, and to contain albumen and some small round masses, consisting of red blood-corpuscles, mucus, and large oval and round cells in an advanced stage of fatty degeneration." On February 23rd the urine was clear, acid, of sp. gr. 1010, and contained a distinct trace of albumen. The tumour had increased in size considerably since February 7th, and had become more nodular. The temperature had been normal throughout. He left the hospital on March 5th, but returned again on the 29th much weaker, with much more pain and with much greater difficulty in passing his water, which was much more albuminous. The tumour had still further increased in size. On April 10th he became unconscious, and died thirty-six hours later.

At the autopsy the tumour was found to weigh 6 lbs. 2 oz. On section it was seen to be firm and yellowish at the periphery, broken down and hæmorrhagic in the centre. There were no remains of unaltered kidney tissue to be found, and the left supra-renal body did not appear to be incorporated in, or to be the starting-point of the new growth. The tumour was entirely surrounded by a thin capsule. The left ureter was much dilated and hypertrophied, being 1¼ inches in external diameter. Its opening into the bladder allowed the passage of a large probe easily. The bladder was enlarged. Lying loose in its cavity was a small fleshy-looking mass, about the size of a hazel-nut, which was encrusted with phosphates. This mass

proved on microscopical examination to consist almost entirely of large round sarcoma cells. From the neck of the bladder there grew a similar mass. The urethra was healthy; there were deposits of new growth in the retro-peritoneal glands and in the right lung, otherwise the organs of the body appeared to be healthy. Unfortunately the brain and eyes were not examined. In conclusion I have to thank Mr. Bennett for permission to bring the specimens before the Society, and to thank Dr. Delépine and Mr. Bull for allowing me to make use of their notes.

December 20th, 1892.

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2. *Primary carcinoma of the kidney.* (*Card specimen.*)

By Cecil F. Beadles.

The tumour, a recent specimen, has been divided longitudinally; its weight is 69½ oz. and its total length is 9 inches.

The lower part of the tumour consists of a nearly spherical mass of new growth, measuring 6 inches in diameter from above downwards and 4½ inches across. It is very soft in consistence, and in places is breaking down, and cavities exist filled with fœtid and blood-stained fluid. The general colour is yellowish white, but hæmorrhages have occurred in places. Above this mass is situated the remains of the kidney, which is studded with small deposits of growth of the same nature as that below. The kidney substance is pale and somewhat soft.

Post-mortem fifteen hours after death.

Head: No sign of injury was discovered, and the calvaria was normal in appearance. The pio-arachnoid membrane was greatly thickened and opaque, especially over the upper part of the brain, and there was a considerable amount of fluid within the skull. The brain weighed 50½ oz., was pale and anæmic, and firmer than natural; the ventricles were not dilated, and there were no signs of old hæmorrhage. The arteries at the base were fairly healthy.

Thorax: Much fluid existed in the pericardial cavity, and the heart was covered with rough lymph. The heart was large and

hypertrophied, especially the left ventricle; weight 16½ oz. The mitral and tricuspid orifices were dilated. The aortic cusps were atheromatous and very calcified, as was also the first part of the aorta to a slight extent.

Lungs: The left was somewhat collapsed, and the base of the right was congested. No fluid existed in either pleural cavity, and there were no adhesions.

Abdomen: On opening the abdomen a large tumour was seen to occupy the right side, projecting forwards from beneath the liver; its anterior surface was smooth and round. This was found to be the tumour already described, which was united to the right kidney, from which it was inseparable. The tumour was quite adherent to the lower and posterior part of the right lobe of the liver, and had to be cut away, and the growth was firmly adherent to the abdominal wall behind, and was torn through in removing it. The liver was of great size, weighing 77½ oz., very pale, soft, and fatty, and was flattened out anteriorly, where it overlapped the smooth surface of the tumour. There were a few nodules of growth of a pale colour situated in the liver, where its adhesion to the tumour existed posteriorly, due apparently to direct extension. Beyond these no other secondary growths were found in the body.

Left kidney was large, soft, pale, and fatty. The capsule was adherent in places, and it weighed 6½ oz. Spleen large and soft, measuring 7 inches in length and weighing 12 oz. Bladder healthy.

History.—The specimen is from a man (10846) aged 57, who had been an inmate of Colney Hatch Asylum since February 10th, 1890. On admission he was the subject of epilepsy with aural hallucinations, and the following note was then made:—"A fairly nourished man of middle age. He states that he served in the army for fifteen years, and that for nine years of that time he was stationed in India; his first fit occurred about two years ago. He is quiet and well-behaved, and answers questions with fair intelligence, but his memory as to recent events appears to be defective."

April 6th, 1890.—Had three fits last night, the first he has had since admission.

January 24th, 1891.—Is usually very well behaved and quiet in manner, but when his fits are coming on he is vacant and irritable. These he has occasionally. Health is fair, and he is usefully occupied.

January 14th, 1892.—Somewhat weak-minded, and at times irritable and quarrelsome. He is not known to have had a fit for more than a year.

On June 23rd, 1892, he had a quarrel with another patient, and during a scuffle which followed he had a fall. Since this time he has never felt well, and he gradually got worse as time went on. In February last he was complaining of feeling weak with pains in the loins, more especially on the right side. He was very thin, and his legs were sometimes swollen. Urine contained a considerable amount of albumen, and since the accident mentioned above there had been a small amount of blood occasionally in the urine at intervals of several weeks.

On April 12th, 1893, he was much worse, with constant pain in the back. There had been no hæmaturia since the last note.

April 17th.—Last few days has had vomiting and severe pains in his chest, which passed through to the back. He died in the evening.

It is interesting to note that lately it had been ascertained that this patient served in the Enniskillens during the Crimean war, and was apparently one of the few survivors of the "600 heavy brigade" who charged at Balaclava.

There was a strong history of neoplasms in the family. A relation of the patient states that his mother died in Victoria Park Infirmary with two tumours, that a sister died in St. Bartholomew's Hospital who had three tumours, and that another sister has been supposed to be suffering from a tumour for the last eleven months.

Microscopical examination.—Sections were cut from several parts of the growth and stained with logwood. From these it appeared that the greater mass of the tumour was in a necrotic state, but here and there a fine alveolar structure was seen, the meshes being occupied by cells in a more or less granular necrosed condition, comparatively few of them taking the stain properly.

A section of the kidney substance showed the changes observed in a "mixed kidney."

April 18th, 1893.

3. *A calculus encysted in a child's bladder.* (*Card specimen.*)

By D'Arcy Power, for Guy Neville Stephen, Government Medical Officer, Cyprus.

Mr. Stephen thinks that this specimen is worthy of exhibition for the following reasons:

1. It is an example of a stone which could not be removed by supra-pubic lithotomy, but which could readily have been extracted after perinæal section.

2. It gave rise to intense and continuous agony, although encysted calculi are usually comparatively painless.

3. The stone and the cyst are very large in comparison with the bladder, which is greatly hypertrophied and contracted.

4. The shape and position of the stone are peculiar. The calculus runs right under the prostatic and membranous urethra, and is enclosed in a cyst which is not merely a protrusion of the mucous membrane, but is a true sacculation of the bladder wall.

The patient was a wasted boy, said to be six years of age, whose life had been a protracted agony for two years. His hands were excoriated from the constant passage of urine over them as he held his penis during micturition.

The bladder was opened, under chloroform, above the pubes, when it was discovered that the projecting beak of the stone presented high above the floor of the bladder. This projecting portion broke off during manipulation, and it then became evident that the rest of the stone could not be removed by the supra-pubic incision, as a fine probe passed through the opening into the cyst showed that the calculus was very much larger than the neck.

The child died twenty-four hours later. *November*, 1892.

4. *Casts from a case of membranous desquamative urethritis.* (*Card specimen.*)

By William Henry Battle.

A series of casts were shown corresponding to the diameter of the urethra, and of varying length, some as long as three or four inches, the majority short, with shreddy ends. They were wrinkled, semi-translucent in parts, and whitish, with yellowish areas, apparently due to increased thickness. They form almost perfect tubules of a diameter corresponding to that of the urethra.

Microscopically they consist of large epithelial cells, with many leucocytes, a perfect membrane being formed.

From a man aged 45, recently under care at St. Thomas's Hospital.

They are interesting as representing the morbid products of a disease not hitherto described in this country.

May 16*th*, 1893.

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5. *Calcareous plate removed from the dorsum of the penis.*

By Edgar Willett.

In January, 1891, a clergyman, aged 51, asked advice for trouble resulting from a hard mass just beneath the skin of the dorsum of the penis: it had existed for two and a half years, but had increased quickly during the last two months; during erection the penis became curved backwards. It formed a kind of plaque, felt like cartilage, and had curved edges; it lay just beneath the skin, and was removed without difficulty. It was close to the pubic bone, but was not attached to it. It seemed to have arisen in a part of the fibrous tunic of the penis. The patient made an excellent

recovery. When examined more carefully after removal, it felt hard, like bone, and could not be cut with a knife. A microscopic section, prepared by softening a small portion in hydrochloric acid, showed that the structure was calcareous rather than real bone.

The case occurred in the practice of Mr. Butlin.

October 18*th*, 1892.

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6. *Localised thickening in the corpus cavernosum.*

By Sydney Jones, M.B.

T. B. B—, aged about 45, came to Mr. Sydney Jones on August 30th, 1892, with a fibrous thickening on the dorsum of the penis, about one inch below its root. This thickening was round, shaped like a shilling, and about the size of that coin. The skin was perfectly free from it, and it could be lifted with the finger and thumb, so as to seem isolated from the corpora cavernosa, although its deep connections were no doubt involved in these bodies.

There was no history of syphilis. The skin was uninvolved; there was no sore nor induration on the glans below, and no glands enlarged in groins.

He had noticed the thickening some few weeks, he did not know how long. His ideas were set on cancer, his grandfather having died of cancer. I did not expect to have much influence upon it, but prescribed iodide of mercury and Ung. Pot. Iodid. I told him decidedly that it was not cancer, but felt that in my experience I had not come across anything like it, and would be glad to know the experience of the Members of this Society.

This gentleman's state of mind was such, from the prevailing idea of cancer, that I considered it expedient to advise this fibrous thickening to be removed, and this I did on the 6th of September, exactly one week after he first consulted me.

Matters have gone on very well since the operation, the healing being disturbed occasionally by erections and consequent slight hæmorrhages.

Report by Mr. Shattock.—"The tissue removed consists of a portion of the corpus cavernosum, one by three quarters of an inch in superficial area, and half an inch in its extreme thickness. There is nothing histologically abnormal in the microscopic section, *i. e.* the tissue elements, fibrous and muscular, are natural, the plain muscular tissue in the trabeculæ being especially noteworthy in amount. The bundles of this tissue are quite easily distinguishable from the connective tissue of the trabeculæ, in which tissue they lie; for not only do they present the typical long rod-shaped nuclei, both in longitudinal and transverse sections, but the cell body is, in addition, lightly stained throughout with the hæmatoxylin (Erhlich's), whilst the bundles of connective tissue are quite devoid of colour.

"The thickness of the fibrous investment, as measured in the microscopic section, is 3 mm., the extreme thickness of the subjacent cavernous tissue is 6 mm. As compared with sections of the normal organ the investment is unnaturally thick, and the trabeculæ are markedly so, the cavernous structure being very obviously, even to the naked eye, much closer than normal in the same situation. In the fibrous investment at one spot is a small plate of dense calcareous-looking substance: this, on analysis (by Professor Dunstan), proved to be phosphate of magnesia and lime, with no trace of uric acid. A gouty ætiology of the lesion may be thus precluded." *October* 18*th*, 1892.

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7. *Primary sarcoma of corpus cavernosum penis.* (*Card specimen.*)

By William Henry Battle.

The specimen shows portions of both corpora cavernosa, with part of the penis after their junction. To these is attached an ovoid tumour, measuring about four inches by two or more inches in width, growing almost exclusively from the under surface of the organ, and especially from the left corpus cavernosum. It

also extends into the right crus, at its junction with the left, and invades the body of the penis. It consists of soft growth, with cavities where myxomatous changes have taken place. It is provided with a fairly well-defined capsule.

Microscopically, as shown by the preparations, it consists of large-celled mixed growth, undergoing myxomatous degeneration.

Removed, January 30th, 1893, from a patient aged 30, an inmate of the Royal Free Hospital. He had noticed a swelling in the perinæum for two years, and when admitted this was the size of a fist, and very elastic. There were no enlarged glands. The anterior part of the penis was not removed.

The man made a good recovery, but at the time of exhibiting the specimen is still troubled with a small urinary fistula in the perinæum. *May 16th*, 1893.

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8. *Extensive rapidly destructive papilloma of the penis.*

By W. Arbuthnot Lane, M.S.

W. J. L—, aged 39, was admitted into Guy's Hospital, under my care, on September 1st, 1892. He had never before suffered from any venereal complaint. He found his foreskin swollen about four months ago. This occurred three weeks after connection. Dr. T. Parker, who attended him, states that when he first saw him the penis was swollen, and that there appeared to be a ragged chancre on the lower margin of the prepuce, which soon became phagedænic. Nitric acid and charcoal poultices were applied with only transient improvement. Ulceration and profuse hæmorrhage with much pain and restlessness ensued. Though everything was done that could be done, the organ continued to enlarge.

On admission, the body of the penis was represented by a large, soft, globular mass, which measured about 3 inches in its several diameters. The surface of this growth, which was very soft, was obviously warty. The margin of the glans was affected, and posteriorly it terminated abruptly at the root of the penis. Within the orifice of the urethra some growth could be seen. He was

treated with inunctions of oleate of mercury, mercurial baths, &c., with no benefit.

On September 3rd, the patient being under an anæsthetic, the growth was examined, when it was found to present the same character throughout, namely, a very soft, branching, warty structure. On attempting to shave the bulk of the tumour off the organ, it was found to penetrate it everywhere, especially the corpus spongiosum, where the urethra was opened up for nearly a couple of inches.

The inner surface of the urethra beyond this limit was lined with the same soft warty growth.

The appearances presented by sections of the tumour suggested that the growth had commenced in the urethra at no great distance from its orifice, and that it had destroyed the floor of the spongy body and prepuce, where it simulated at first the appearance of a phagedænic chancre. It then extended over the surface of the corpora cavernosa and spongiosum, invading and destroying their capsules.

Finding that it was hopeless to attempt to save the organ, it was amputated immediately behind the limit of the growth, where the section of the urethra in the stump was found to be free from warty formation. The man made an uninterrupted recovery, and no recurrence of the growth took place.

Sections of the tumour showed that it consisted throughout of the same papillomatous structure, there being no evidence of any epithelial ingrowth, ulceration, or sloughing. The deeper portion of the growth simulated a sarcoma, both in structure and in the manner in which it infiltrated the several fibrous structures forming the body of the penis. *December 20th*, 1892.

Report of the Morbid Growths Committee upon Mr. Lane's specimen of destructive papilloma of the penis.—We examined transverse sections of two parts of the penis affected by the growth, and a longitudinal section through the glans penis and urethra. The latter specimen showed a papillary growth springing from the epithelial lining of the urethra, and continuous with a similar papillomatous condition of the epithelial covering of the glans. The papillæ growing from the surface of the urethra consisted of a central stalk of vascular connective tissue, which, at its periphery, broke up into numerous branches. These were covered by several layers of densely-packed

epithelial cells, which under a high power were seen to be of various forms of transitional epithelium similar to those lining the deeper part of the normal urethra. In this portion of the penis the growth was purely papillomatous, and showed no tendency to infiltrate. An examination of the transverse sections of the deeper parts of the penis shows that the greater portions of the corpora cavernosa and of the corpus spongiosum have been replaced by the new growth. This consists of a stroma composed of connective tissue, rich in cells, and arranged in the same papillary manner as in the previous section. This connective-tissue framework was covered by numerous layers of epithelial cells of the same type as those which covered the papillæ in the urethra. The papillary processes in this part of the tumour appeared to have coalesced in places, and had thus formed alveoli, which contained masses of epithelial cells. Throughout this part of the specimen the tumour had infiltrated and replaced all the adjacent tissues. The projecting papillary masses, described by Mr. Lane, consisted of a similar structure. With regard to the stroma of the growth, we should regard the cells alluded to by Mr. Lane as irritative rather than sarcomatous in origin. The tumour appears to be an infiltrating epithelial growth, and from the nature of its cells it has evidently originated from the urethra. In its histological structure it closely resembles "duct carcinoma" of the breast, and, like the latter, should be included in the class of "villous carcinoma."

G. Newton Pitt
Anthony A. Bowlby.
Seymour J. Sharkey.

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9. *Congenital cystic disease of the scrotum.*

By Edgar Willett.

Two specimens were shown of this disease, the chief interest of which lay in its great rarity. One of the cases was under Mr. Smith, and was the smaller of the two; it occurred on the left side of the scrotum of a boy aged 9; it was composed of a number of small cysts, varying in size from that of a hazel-nut to that of a small pea, held together by fibrous tissue; when fresh these were

soft and translucent. Although it was in close proximity to the testis, the gland was not affected, and the tumour was dissected out without injury to it. The tumour was noticed shortly after birth, since which time it had slowly increased.

The second case occurred also in St. Bartholomew's Hospital, and was under the care of Mr. Marsh; it was rather larger, but was very similar in appearance both before and after removal, and after hardening and cutting, to the other specimen. The boy in this case was aged 11, and the tumour also had been noticed and had slowly increased since birth: it had rather deeper connections than the other one, and seemed to be attached to the pubes. It occurred on the right side.

The tumours consisted of a series of cysts, when fresh containing a clear fluid, which was coagulated by the spirit in which the specimens were hardened, and having a very fine lining membrane.

The solid parts were composed of fibrous and connective tissue, and scattered amongst this there occurred here and there bundles of unstriped muscular fibres. Microscopical sections were shown which confirmed this.

The pathology of the tumours is obscure: similar ones occurring either in the scrotum or in other parts of the body have been recorded. At the Royal College of Surgeons there are no tumours like these from the scrotum, but there are three others somewhat similar in appearance. One (No. 302) is a multilocular cystic tumour removed from the thigh of a little girl aged 4. Another (No. 303) was removed from the gluteal region of a boy of 8½; and the third, one having a much more defined outline (No. 303A), was removed from the left hypochondrium of a girl aged 15.

Accounts of two or three others which occurred in the scrotum may be mentioned. One of these is recorded in the 'Dublin Hospital Reports' for 1857 by Fleming, and the other by Curling in his book on the testis. Both occurred in boys.

The French surgeon Dolbeau also mentions a case, which was in all probability similar to those now shown. The patient was a little boy aged 1½. Another French surgeon, Lannelongue, in discussing the origin of these curious growths, suggests that they may be formed in the inclusion of a part of the Wolffian body. Those in the Royal College of Surgeons, which have already been alluded to, are described in the catalogue as probably being of the same nature as the congenital cystic hygromas of the neck. These tumours, it

is known, are not confined to the neck. Two cases have occurred in the arm, and one over the region of the right hypochondrium.

In vol. xxxvi of the 'Transactions' a congenital tumour of the perinæum is described by Mr. Turner, which may have been of the same nature. *October* 18*th*, 1892.

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10. ***Carcinomatous growths in parietal layer of tunica vaginalis secondary to primary vesical growth; ? implantation through unobliterated funicular process of tunica vaginalis.*** (*Card specimen.*)

By H. D. Rolleston, M.D.

The left spermatic cord, tunica vaginalis, and testis are shown. The tunica vaginalis has been opened. The parietal layer has flat masses of new growth on its inner surface. There is no growth on the visceral layer, or in the cord (see Fig. 6).

Fig. 6.

Above the testicle is the epididymis. The sac is laid open to show the nodular new growth. The funicular process is patent and of considerable size, as shown in the cut surface of the cord.

The testis was removed by pulling on the vas deferens inside the abdomen. Unfortunately it was not noted before this was done whether the funicular process of peritoneum was patent at the internal abdominal ring. It is patent, however, about as far as the external ring, that is, in the portion of cord preserved.

The specimen was removed from the body of a man aged 44, who died with an extensive rapidly growing carcinoma of the fundus of the urinary bladder. There were secondary growths in the peritoneal cavity.

It seems probable that pieces of the primary growth becoming dislodged passed into the patent funicular process of tunica vaginalis and became implanted on the inner surface of the parietal layer. Unless it did occur in this way it is hard to imagine how the growths in this situation were brought about, as the cord is quite free. Microscopically the growth in the tunica vaginalis resembled the primary carcinoma in the bladder. For the drawing I am indebted to Dr. G. H. Goldsmith.

May 16*th*, 1893.

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11. *Medullary sarcoma of both ovaries and of the peritoneum in a child aged* 6. (*Card specimen.*)

By F. C. Turner, M.D.

The specimen consists of the pelvic organs of a female child aged 6. Both ovaries are converted into rounded masses of medullary sarcomatous growth. The right ovary is as large as a full-sized orange. The left ovary is smaller; it was adherent to a large mass of growth surrounding the cæcum and commencement of the ascending colon. The right ovary was free. There were, moreover, nodules of subperitoneal growth in the mesentery contiguous to the intestine, and some of the appendices epiploicæ were converted into masses of growth. The bowel was not invaded by it, nor were growths found in any other organs.

Sections of the growth in the ovaries showed the structure of small round-celled sarcoma. No muscular fibres were discoverable.

The child from whom the specimen was obtained had always been delicate, and had had measles, whooping-cough, and influenza.

She was admitted into the London Hospital in July, 1892, having

been losing flesh and strength since the previous March, with abdominal distension and difficulty with the action of the bowels. She then appeared to be suffering from tubercular peritonitis with adhesions of the bowels, without any exudation being discoverable. Subsequently the presence of tumours first on the right, and later on the left side of the abdomen became evident. During September and October these rapidly developed, and the patient became very wasted. The abdomen became distended and the respiration embarrassed. She died on November 15th. No enlargement of the subcutaneous glands occurred.

January 17th, 1893.

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12. *Papillomatous tumour of both ovaries.*

By P. H. Pye-Smith, M.D., F.R.S.

These two large masses of villous or papillary growths, affecting both ovaries nearly equally, were taken from the body of a patient who had been for nearly nine years under my observation, and during that time had been tapped no less than 299 times "without once complaining of her lot."

In August, 1884, she found her abdomen swelling, and on the 20th of September following came into Guy's Hospital under my care. She was then a healthy married woman of 35, with four children. There was a moderate amount of ascites without any evidence of ovarian disease, and with no symptom of cirrhosis. The other organs were normal, and I had paracentesis performed without any decided opinion as to the cause of the peritoneal effusion. On this occasion, the first of nearly 300 similar operations, nine pints and a half of clear yellow serum was drawn off, reducing the circumference at the umbilicus from 39 to 30 inches. No tumour was felt afterwards, and the uterus and other pelvic organs appeared to be normal.

She was readmitted on November 10th (six weeks after the first tapping), and was relieved a second time. The ascitic fluid measured twenty-four pints, and was of the same negative character.

The circumference of the abdomen was reduced from 42 to 35 inches.

At the third operation on December 19th, the abdomen measured 47 inches; and, after removal of twenty-eight pints of serum, 40 inches.

During the year 1885 she came in seven times to be tapped, and continued otherwise in good health, with no fresh evidence as to the cause of the disease. At the eighth tapping, in July, twenty-one pints were drawn off, and the serum was for the first time stained with blood. On this occasion the microscope for the first time showed several large epithelial cells, and fulness was felt in the pelvis, which made me suspect that the disease might be colloid, or some other form of pelvic and peritoneal cancer. The recti had become separated by pressure, and there was prolapsus of the hinder wall of the vagina. The next time, however, the serum was clear from blood; and it did not seem justifiable to urge more strongly the advantage of an exploratory operation, which had been already proposed to the patient but declined. The twenty-first tapping was in December of this year.

During 1886 paracentesis was performed eleven times. The patient's health continued excellent. The amount of serum drawn off was usually less than in 1885, varying from twelve to twenty-three pints. In May it is noted that seventeen pints was the amount removed; the serum was of a greenish-yellow tint, sp. gr. 1030, and alkaline. It slowly formed a loose coagulum after standing, and became nearly solid on boiling. It contained the ordinary salts, without sugar or bile-pigment, and showed only traces of blood under the microscope. Chlorides were abundant, and there was a trace of urea. In August of this year a distinct tumour in the right iliac region was detected. In December it was thought that both ovaries could be felt prolapsed by the side of the uterus, and this appeared to confirm my belief that the disease was colloid. In February, 1887, the patient was again urged and at last persuaded to undergo an exploratory operation. On the 24th the abdomen was opened, and eighteen pints of clear yellow serum removed. A multitude of translucent gelatinous bodies were seen studding the parietal and visceral peritoneum, and the diagnosis I had formed of colloid cancer seemed to be verified. I had, however, put a finger through the small exploratory opening, and afterwards found a minute fragment of tissue under the nail, which

showed a villous structure. The patient's reluctance to submit to surgical treatment was unfortunately confirmed. During 1887 paracentesis was performed eighteen or nineteen times.

In March, 1888, Mrs. S— attained what she called her "jubilee," but without any sign of release from her disease. The tappings between the fiftieth and the one-hundreth became more frequent, and I repeatedly found in the deposit from the serum not only epithelial cells but villous processes, so that the conclusion was clear that there was no colloid growth of the peritoneum, but papilloma of one or both ovaries with secondary peritoneal colonies. I advised a second attempt to remove the source of the disease, but this was steadily refused, or at least postponed, and I must confess that her general health continuing so good, I did not perhaps urge an operation so persistently as I should.

During 1889 and 1890 the case presented no new features, except that the intervals between the tappings became still shorter and the masses filling up the abdomen became gradually larger. In March, 1889, I again proposed an operation, but again unsuccessfully. The patient ate, slept, and looked well; and, except from the pressure of the fluid a day or two before paracentesis, professed to suffer nothing. In June, however, she had some decided abdominal pain and vomiting, which were at once relieved when thirteen pints of blood-stained serum were removed (ninety-first paracentesis).

In 1891 she was tapped almost every week, the amount of serum removed being usually between seven and ten pints. We frequently noticed white flocculi in the serum, visible to the naked eye or at least forming a visible deposit. These showed the most beautiful villous structure. The epithelial cells were club-shaped or cylindrical, with clear protoplasm and large oval nucleus. They formed only a single layer, and rested on very delicate connective tissue with blood-vessels. When looked at on the surface instead of in (optical) longitudinal section the free extremities of the cells formed a regular pavement of more or less hexagonal elements.

In June, 1892, Mrs. S— was tapped for the 245th time. During the whole year there was no change in the condition of the patient or in the symptoms of her disease.

In January, 1893, the 277th tapping showed abundant white flocculi, some just visible, others as large as a pea: they all consisted of villi like those before described. The serum amounted to

four pints, and was free from blood. During February and March Mrs. S— was tapped ten or eleven times, and again on the 6th, 11th, 18th, and 21st of April. Three days later (April 24th), she came in for the last time with more pain and distress than ever before; and she was not relieved as on previous occasions, after five pints of serum had been drawn off. On the contrary, she suffered from severe abdominal pain and persistent vomiting. It was thought that acute, perhaps suppurative, peritonitis had ended her life.

At the *autopsy* (April 26th) there was fresh accumulation of blood-stained serum, but no trace was found of pus or recent lymph. The abdominal mass, which filled the pelvis and rose to within an inch of the umbilicus, was seen to consist of both ovaries. Each of them was transformed into a huge tumour, looking like the head of a cauliflower and consisting of long branching villous processes, some dead-white, others pink, and some dark purple from congestion. The right was the larger, and rose higher in the abdomen, as was ascertained during life. Its diameter was about 4½ inches, that of the left tumour about 3½. The uterus was pushed up between them out of the true pelvis. The continued morbid growth had redressed the procidentia uteri which it had once caused, first pulling on the broad ligaments, and then squeezing the uterus upwards in the direction of least resistance; for the pelvis was quite full.

The peritoneum covering the anterior parietes of the abdomen was greatly thickened, opaque, and covered with secondary papillomata. Here extensive adhesions had taken place to the uterus as well as to the ovarian tumours. The omentum was thick and contracted. The visceral peritoneum was for the most part free, but there were several secondary villous growths scattered over the coils of the small intestine.

The abdominal viscera beside the ovaries were all of normal structure; but the liver was firmly adherent to the diaphragm and stomach, and the spleen was adherent also; the tumour was firmly connected to the sigmoid flexure of the colon, and to the anterior parietal peritoneum. In particular there was no sign of dilatation of the cæcum or colon from pressure on the rectum, nor of pyelitis, hydronephrosis, or chronic Bright's disease from dragging on the ureters.

The heart and lungs were also perfectly normal. In fact, with

the exception of scars in the vagina and the cervix uteri, the ovaries and peritoneum were the only diseased parts.

The immediate cause of death was syncope from uncontrollable vomiting, but why this had not occurred at a previous period of her illness is hard to say.

My diagnosis in the earlier stages of the disease vacillated between colloid cancer and multiple papillomata of the ovaries. The recognition of the villous processes under the microscope made the existence of the latter condition certain.[1]

It would have been better practice to have urged ovariotomy more strongly; and the operation would have been possible, though hazardous, even after adhesions had formed. In similar cases it has been found that the secondary growths often wither and disappear when the primary disease has been removed.

It is clear that the disease of which this patient died is not in any proper sense of the word "malignant;" it does not invade tissues, nor reappear in distant parts; and it only caused death after a course of nearly ten years, and that by reflex irritation such as any ovarian disturbance may give rise to. The villous form of the tumour, like that of tumours of the bladder, renal pelvis, and occasionally of the stomach or intestine, probably depends on free growth taking place in a liquid medium; but why all tumours growing in ascitic fluid or in urine do not assume the same branching papillomatous habit is not evident. I supposed that the growth was originally cystic—an example of what used to be described as a multiple proliferating cystic tumour of the ovary—comparable to the cystic adenocele of the mammary gland. And there can be no doubt that such papillomatous growths may arise in the cysts of an ovarian tumour. But anatomically, I found no trace of cysts after death, and it is probable that in this as in some other cases the papillomata grew out, not from the lining of a cyst wall, but from the free surface of the epoöphoron (parovarium, or organ of Rosenmüller). In either case the propagation of the disease obviously takes place by detachment of fragments of the ovarian tumour and their becoming engrafted on the visceral or parietal peritoneum wherever they may chance to lodge.

[1] On one occasion, three or four years before her death, Mrs. S. had come in with a clear account of having brought blood up from the stomach. She was a publican's wife, and with ascites and hæmatemesis was naturally supposed by those who did not know her case to be suffering from cirrhosis of the liver.

The characters of the ascitic fluid do not afford any sure means of distinguishing between the passive effusion due to cardiac or hepatic disease, the more or less decidedly inflammatory exudation of renal dropsy, and that poured out in chronic peritonitis, whether "idiopathic" or due to the presence of a new growth.[1]

The presence of blood, though most frequent in cases of carcinoma, may be met with whenever a growth is vascular, as in the present case. Clots often form when the ascites is by every accepted rule, a strictly passive, dropsical effusion. A clear watery colourless liquid indicates a parovarian cyst, but in this case the primary disease, if parovarian in origin, was not cystic, and the ascites was caused by peritoneal irritation. A thick gelatinous fluid may have been formed in an ovarian cyst, or it may be derived from colloid cancer of the peritoneum, and in either case it may have a deep yellow-brown or greenish colour.

The imperfect evidence, however, which was furnished by the characters of the ascitic fluid was in this case supplied by microscopic examination, the presence of the minute flocculi above described. Their characteristic anatomical structure was decisive of the nature and seat of the disease.

The size of the tumours, the long duration and complete history of the case, and the important bearing which its pathology has upon the prognosis and treatment of similar papillomatous growths, seemed to justify its being brought before the Society.

By a remarkable coincidence, another patient died in the same month as Mrs. S—, who had also been repeatedly under my care in Guy's Hospital, who had also been repeatedly tapped and whose case offered the same difficulty of diagnosis between ovarian disease and colloid cancer of the peritoneum. The fame of Mrs. H—, a respectable married woman of 54 sent up to me by Dr. J. M. Hobson of Croydon, in November, 1892, was eclipsed by that of Mrs. S—, but she attained the not inconsiderable distinction of being tapped 36 times.

The case was at first regarded as one of cystic disease of an

[1] An examination of the ascitic fluid made by Mr. F. G. Hopkins (Gull Student in Pathology) showed that there were present serum-albumen and globulin, with small quantities of fibrinogen and of normal mucin, but none of the so-called pseudo-mucin found in ovarian cysts. It was always coagulable by heat, and usually formed a loose clot after standing. It contained the usual salts of serum.

ovary setting up secondary ascites. The fluid drawn off by paracentesis was usually ascitic (straw-coloured serum with a greenish tinge, of rather low sp. gr., 1010 to 1015, containing albumen, but not clotting and showing no formed elements under the microscope), occasionally thick, gelatinous, brown or olive coloured, like that often furnished by ovarian cysts.

As in the other case, an exploratory operation was undertaken, but the peritoneum was found thickened with colloid growths and nothing further was attempted, beyond repeated paracentesis.

Latterly, the intervals between her visits to the hospital to be tapped became very short, and there was constant difficulty in evacuating through a cannula the thick, jelly-like, "colloid" material. The microscope occasionally showed a few nucleated cells larger than leucocytes but not epithelioid in character.

After death (November 19th, 1892), the peritoneum was found to be pervaded by colloid growth. Its primary seat was not ascertained, but from the course of the symptoms it most likely, I think, began in the pelvis. There was only one secondary deposit, a nodule in the liver. The ovaries were normal. *May 2nd*, 1893.

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13. *Tuberculosis of the Fallopian tubes, uterus, and vagina.*

By CHARLES A. MORTON.

THIS specimen of tuberculosis of the generative tract was removed, *post-mortem*, from a girl, 8 years of age, who died of phthisis. There was some dry tubercular peritonitis; and all the parts in the pelvis, small intestine, and uterus and appendages, and bladder and rectum, were matted together. On each side of the uterus, caseous breaking-down masses, the size of walnuts, were seen. These were supposed to be tubercular ovaries, and one was, with much difficulty, dissected out of the adherent mass attached to the side of the uterus. The interior of the uterus was found to be nodular and ulcerated, and the upper portion of the vagina was similarly affected. After hardening, a section was carried through the outgrowth at the side of the uterus, and this was found to be not an ovary, but a growth formed of enormously thickened folds of the lining membrane of the tube, looking like a microscopic section

of this structure, with its folds cut transversely. It was round, and measured an inch in diameter, and the wall of the sac containing the thickened processes was itself of considerable thickness. The tube leading from it into the uterus was patent. It was found that the ovaries and the other tube had, unfortunately, not been successfully removed from the adherent mass in the pelvis.

Microscopic sections of the growth in the right tube show that it is composed of very marked cellular infiltration of the folds of lining membrane of the tube. Only one or two imperfect nodules of cells could be made out, and no giant cells were seen, and thus it was feared that it would be difficult to demonstrate its tubercular nature, though its association with tubercle elsewhere made this highly probable. However, an enormous number of tubercle bacilli are to be seen in it.

Secondary tuberculosis of the female generative organs is not very rare, but few cases have been placed on record in this country, and the only specimens exhibited at this Society seem to be those of Dr. W. S. A. Griffiths and Mr. Walter Edmunds, in 1888 ('Trans.,' 1889) and Dr. Bristow as long ago as 1855. Since the communications to the Society on this subject in 1888, a very interesting case of tuberculosis of the tubes and uterus (in which the ovaries were not tubercular), associated with tubercle in the brain, has been placed on record by Dr. Ballantyne and Dr. J. D. Williams of Edinburgh,[1] and a paper of much value has recently been published in the 'Pathological Reports of the Johns Hopkins Hospital,' by Dr. Whitridge Williams, on an examination of nine specimens, with reference to 133 papers on the subject. He has come to the conclusion that it is usually secondary to tuberculosis elsewhere, but in a considerable number of cases is primary; and that many tubes which are removed by operation and thought to be affected merely with salpingitis, are on careful microscopic examination, found to be tuberculous; a condition which Dr. Griffiths thought probable, when in his paper in the 'Transactions' of this Society in 1889 he asked the question, "May not the so-called idiopathic cases of suppurative salpingitis in girls and young women who are beyond suspicion of gonorrhœa, or similar causes, be due sometimes to this?"

May, 2nd, 1893.

[1] 'Brit. Med. Journ.,' 1891, vol. i, p. 168.

14. *Sarcoma of the uterus, showing extreme cystic degeneration.*

By C. J. Cullingworth, M.D.

The specimen consisted of the uterus and appendages removed on the 28th of April from a childless widow, aged 37, by the operation of abdominal hysterectomy. The uterus at the time of the operation was much larger than an ordinary pregnant uterus at full term, but as above three pints of serous fluid escaped from the "tumour" during its removal, the present size of the specimen is much below that. The "tumour" is situated in the substance of the uterine wall, to the left and in front of the uterine cavity. It forms a spherical mass, 6½ inches to 8 inches in diameter. The uterine cavity itself is enlarged, measuring 6½ inches in length by 3 inches in width at the widest parts, the walls being in apposition as usual.

The specimen shows, at its lower end, a circular incision through the entire thickness of the uterine wall down to the capsule of the tumour, the lower portion of which, having been enucleated from the lower uterine segment, now projects in the form of a convex mass below the level of the circular incision.[1]

The tumour admits of being enucleated throughout. It is then found to have a comparatively thin wall of fibrous tissue, and to consist, internally, almost exclusively of serous-looking fluid, contained in a coarse meshwork of delicate, easily-torn, membraniform connective tissue, of a pale yellow colour, in which vessels full of blood are seen, and knots of harder substance, like that to be described later on. When the whole of the fluid has drained away, the tissues mentioned are, with the exception of the tougher fibrous capsule that encloses the whole "tumour," and some discoid formations in this capsule (to be next described), the only solid structures that remain. The flattened, somewhat lenticular masses just mentioned, of which the largest is about equal in diameter to the width of a man's palm, occur in a few places in con-

[1] The level of the circular incision was about an inch below that of the upper border of the broad ligaments.

nection with the capsule of the "tumour," and merge on their inner side into the mesh-work, described as forming the main part of the growth. In section, they contain ill-defined cavities, crossed by delicate connective tissue, and filled with fluid like that in the larger spaces on their inner aspect.

It may be concluded, therefore, that the main part of the tumour is due to similar changes having affected what was once a more solid growth.

The ovaries and tubes are normal except that one ovary was the seat of a cyst about equal in size to a small orange.

Report of microscopical appearances, by Mr. Shattock.—Histological examination of the solid peripheral portions of the growth shows it to be composed of intersecting fasciculi of elongated cells without any formed fibre. The nuclei of the cells are throughout large and oval, and show a very distinct intra-nuclear network, whether in longitudinal or transverse view. Some are so short, even when in longitudinal sections of fasciculi, as to be almost spherical. For these reasons, the tumour must be considered a spindle-celled sarcoma, rather than a myoma.

May 2nd, 1893.

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15. *A case of pseudo-hermaphroditism.*

By J. Jackson Clarke, M.B.

A child christened Frances on account of the sex being unknown, died aged 7 years with suppression of urine.

The parts comprised in the specimen, removed after death, were placed in the author's hands by Mr. H. W. Page. They are represented in Fig. 7.

The uterus and vagina are of the perfect female type. The left ovary alone remains attached to the uterus, the right is wanting. Microscopic examination of the remaining ovary shows it to have a perfectly normal structure. There is a perforate penis and urethra with the male characters. The urethra is dilated, save at the

meatus which is very small, admitting only a fine probe. There are a well-developed corpus spongiosum, glans, and ejaculator muscle. The bladder is markedly hypertrophied, and presents an interesting feature in the presence of symmetrical sacculi as large as Tangerine oranges. The sacculi communicate with the cavity by

FIG. 7.

The pelvic organs seen partly in section, partly in surface view; the sphincter muscles diagrammatic.

rounded apertures about ¼ inch in diameter, and at the back part receive the openings of the ureters, which are somewhat dilated. The wall of each sacculus is about ⅛th inch thick, and is composed of fibrous tissue containing sparse muscular fasciculi.

The most interesting feature of the case consists in the presence

of a blind depression about 1 inch in length in the position of the normal female urethra. This depression is of capillary fineness in its deeper half. A section through the finer part showed that the epithelial lining had fallen away, but a papillated fibrous, and, outside that, a circular muscular coat confirm the impression that the structure represents part of the female urethra. Here then is evidence that a considerable portion of the female urethra is formed from a region of the urogenital sinus, which is independent of the part utilised in the formation of the male urethra. Encircling the orifice of the female urethra was a strong sphincter muscle. Since there is no evidence that any testis was present the individual was doubtless a female. The case would fall into Sir James Simpson's group of transverse hermaphroditism.

The specimen has been presented by Mr. H. W. Page to the Museum of the College of Surgeons. *November 15th*, 1892.

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16. *A case of chronic mastitis in a male aged forty-five.*

By William Anderson.

This case is shown as an example of a condition of some physiological and pathological interest; and one, moreover, that has not hitherto been brought under the notice of this Society.

The patient was a rather weakly man of 45 years of age, but free, so far as could be ascertained, from any inherited tendency to disease. When he first came under my notice he had observed an enlargement of the left mamma for about six months, commencing without apparent cause, and gradual and painless in its progress.

On examination the affected gland was found to be about an inch and a half in diameter at its base, hemispherical in form, moderately hard to the touch, and slightly tender. The skin and nipple were unchanged, and the axillary glands were not enlarged.

In spite of local and constitutional treatment the growth slowly increased, and the patient's health began to suffer from the anxiety awakened by his ailment. Under these circumstances it was

determined to remove the gland, and the operation was performed on the 30th of May, last year.

The tumour, on section, was found to consist mainly of new fibrous tissue developed around the glandular elements. Under the microscope the ducts appeared normal and the acini in the usual rudimentary state, but around each duct and group of acini was a distinct area of proliferation, and beyond this bands of concentrically-arranged fibrous tissue; the whole morbid process appearing to start from the gland structures. There was little or no sign of undue vascularity of the part.

The wound healed readily, but it is worthy of note that the scar-tissue underwent marked "cheloid" hypertrophy. The patient remains quite well at the present time, eighteen months after the operation.

Remarks.—Chronic mastitis in the male in later life is not specially referred to in text-books, and it is unmentioned even in Billroth's monograph. The condition, however, is perhaps less rare than might be inferred from this fact, and I have heard of two examples besides my own. It appears to supply a missing link in the pathological analogies of the male and female mammæ. Mastitis neonatorum and the mastitis of puberty are familiar in both sexes, and it is highly probable that cases like that now recorded correspond to the "involutionary" non-suppurative mastitis that occurs in women who have reached their climacteric. The tendencies of the affection, whether towards resolution, cirrhotic contraction, or malignant degeneration, still remain for inquiry.

December 6th, 1892.

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17. *Tubercular tumour from male mamma.* (*Card specimen.*)

By R. G. Hebb, M.D.

G. M—, gas stoker, aged 31, was admitted to Westminster Hospital under Mr. W. G. Spencer, on September 20th, 1892. In the previous January he received a blow on the left breast, and about six months since noticed a lump there, which had

been growing rapidly during the past three weeks. The tumour, which was about the size of a small hen's egg, lay to the inside of the left breast; it was hardish, slightly moveable, not painful, and not adherent to the skin. There was no definite history of syphilis and no result from anti-syphilitic remedies. He had had several attacks of "paroxysmal hæmoglobinuria." His family history was negative.

Microscopical examination of the tumour shows that it is of inflammatory origin, and probably tubercular in nature, though no tubercle bacilli could be demonstrated. It consists of the usual fibrous and fibrosing tissue, indifferent cells, and giant-cells. There is considerable caseous degeneration, in which the muscle is involved. *December 20th*, 1892.

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18. *Fibroma of the male breast.*

By Cecil F. Beadles.

This is a tumour from the breast of a man, which presents the appearance of a *pure fibroma*, that is to say a tumour composed of fibrous and connective tissue in which there are no epithelial elements. The specimen is of interest, not only on account of the comparative rarity of a tumour in the male breast, but also owing to the extreme rarity of a simple fibroid tumour of the mammary gland. As is well known, the tumours once called fibromata of the breast are now known to be adeno-fibromata, as they contain gland tissue, although sometimes this is in very small quantity. Cases of such have been recorded as occurring in the male breast: one has only recently been published in the 'Lancet' (August 13th, 1892, p. 368). Another case, which is probably similar, is mentioned by Gross as having been recorded by Virchow. But true fibroma of the breast is an exceedingly rare condition, in fact I know of no recorded case; I do not think that one has ever been shown at this Society, and I know of no one who, having had considerable experience in the microscopical examination of mam-

mary tumours, has come across such a growth. Gross ('Tumours of the Mammary Gland,' 1880) has a chapter on fibromata or mature connective-tissue tumours, which he separates from adenomata or simple glandular tumours, but he uses the term "fibroma" synonymously with adeno-fibroma, and it would appear that all his cases of fibroma are tumours in which there still exist the degenerated remains of gland acini and ducts. He makes no separate mention of a fibrous tumour such as one finds in the subcutaneous tissue of the skin. It is possible that Gross, and also others, would classify this growth under the sarcomata, and call it a fibro-sarcoma. But except for the large connective-tissue cells found in some parts, it has not the characters of a sarcoma. In commenting on the case of adeno-fibroma already mentioned, the writer in the 'Lancet' says:—"Pure fibroma of the breast is exceedingly rare, hardly ever occurs. During the last three years, all the mammary tumours in Bergmann's clinic have been carefully examined, and during that time, not a single fibroma has been found."

The notes of the case are briefly these:

W. H—, a man aged 34, presented himself at the Cancer Hospital in May, 1891, with a tumour in the left breast which was of four months' duration. There had been no injury or source of irritation. He had had gonorrhœa fourteen years ago. An uncle had died from epithelioma of the tongue. On admission, a tumour the size of a small walnut was felt above, and a little to the outside of the left nipple; it was somewhat moveable, and was not fixed to the skin. It was extremely hard and was not painful or tender. There was no enlargement of the glands in the axilla. The tumour was thought either a scirrhus or possibly a gumma.

On removal, it was found that the growth was situated at the back of the gland, from which it apparently sprang, and lay embedded partly in the pectoral muscle, part of which was removed with it. It was round in form, and was distinctly limited and quite free from the muscle fibres. On section it presented a hard, whitish surface.

Microscopically, the tumour was composed of dense fibrous tissue, in the central part almost hyaline or myxomatous in appearance with only a few cell nuclei, but at the periphery the growth had a more embryonic character, as is often the case with fibromata. Here numerous large, irregular connective-tissue cells existed, and

towards the edge also were a few slit-like spaces, which appeared to be of vascular origin. No trace of gland acini was present throughout the whole tumour. *December 6th*, 1892.

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19. *Pure sarcoma of breast, with marked gland infection.* (*Card specimen.*)

By Charles A. Morton.

Woman, aged 50, was admitted into Bristol General Hospital, under Mr. Dobson, January 8th, 1893, with the following history. Ten months before admission she had received a blow on the left breast. A month after, she noticed a swelling in it, which enlarged until, just before Christmas, it had attained the size of an orange. At this time she fell and struck the breast again. Since this injury it had grown much more rapidly, and the skin had ulcerated. Four times it had bled, the last time to the extent of a pint. Great emaciation had taken place.

On admission she was very thin, but not very anæmic. The left breast was occupied by a mass of growth the size of an infant's head. The whole mass was firm and bossed. There were two nodules specially distinct on its axillary aspect, both firm, one hard and the other (?) tensely fluctuating. The tumour moved freely on the pectoral beneath. On the front inner surface was a black slough, as large as the palm of the hand. The skin around this was red and much hardened, and in parts œdematous. The veins on and around the base of the growth were much enlarged. The nipple was much retracted, not simply buried in the tumour mass, and there was no morbid growth around it. The skin generally was very tightly stretched over the mass, except around the nipple. In the axilla was a firm smooth mass the size of a duck's egg, not tender. The other breast was normal. The liver could not be felt. There were no signs of growth in the lungs.

After removal the tumour presented a uniform white, soft appearance. There were no cysts. The glands in the axilla appeared

infiltrated with the same kind of growth. Microscopic examination showed the growth to be a small round-celled sarcoma. In the gland examined there was round and spindle-celled growth, and much increase of fibrous tissue. The patient died a month after the operation, and glands were found infected in the lower part of the neck. Sections of one of these showed round-celled sarcoma growth. There were no growths in the internal organs.

May 2nd, 1893.

V. DISEASES, ETC., OF THE ORGANS OF MOTION.

1. *Dislocation of head of humerus with fracture at the anatomical neck.*

By H. H. Clutton.

[With Plate I.]

A woman, aged 61, was admitted under my care on February 18th, 1892, with the following history. She was crossing a street a few hours before with a child in her arms, and fell on to her right shoulder. The child was a "fine boy of two years." This weight undoubtedly added to the momentum of the fall, and in trying to save the child she fell more directly upon the point of the shoulder than she would have otherwise done. There were marks of extensive bruising over right shoulder. The elbow was close to the side, and the axis of the humerus parallel with that of the body. But on pressing the fingers deeply beneath the acromion process the head was found to be absent from its proper position. The same empty space could be felt by grasping this part with the thumb and fingers from before backward. At the same time the shaft of the humerus could be freely moved in all directions without moving the scapula. This caused great pain, and she could not move the arm herself.

On examining the axilla the head of the humerus was felt lying loose and moveable in this space and unconnected with the shaft. The smooth, globular, articular surface presented downwards.

As the patient was over sixty years of age. and the tissues much infiltrated with blood, it was thought advisable to wait a few days before proceeding to operation.

On February 26th an incision was made into the axilla, and as soon as the head of the humerus was exposed it was lifted out of

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

DANIELSSON & Co., Photo Print.

DESCRIPTION OF PLATE I,

Illustrating Mr. H. H. Clutton's communication on Dislocation of the Head of Humerus with Fracture at the Anatomical Neck. (Page 128.)

From photographs by Dr. E. C. Bousfield.

Figs. 1 and 2 show Mr. Clutton's specimen;

Figs. 3 and 4, Mr. Croft's, referred to in the same communication.

the wound without the use of the knife or scissors. The finger was introduced and passed readily through a large opening into the joint. The capsule on the distal side felt rough and bony, and on further examination this rough surface was found to be the great tuberosity torn off from the shaft. The finger was easily swept round the upper end of the bone except at the lesser tuberosity which together with its muscle—the subscapularis—was still connected with the shaft of the humerus.

The wound was closed with a continuous catgut suture without providing for drainage, and healed by first intention.

No passive movement was employed, but the patient was encouraged to use her shoulder as much as possible in various ways. On October 26th, 1892—eight months after operation—the following note was made:—"She can put her hand to the back of her head, to her mouth, and on to the top of the opposite shoulder. She can also put her hand behind her back. The shoulder, however, is rather fixed in raising the arm from the side, and the range of movement seems in great measure to be due to the absence of the head of the humerus. There is flattening of the shoulder and a gap to be felt beneath the acromion process."

The specimen, which is now in St. Thomas's Hospital Museum, comprises the entire head of the humerus, the line of fracture following the margin of the articular surface for two thirds of the circumference of the bone, whilst the remaining third has encroached upon the tuberosities to the extent of about $\frac{1}{8}$ of an inch from the articular margin (see Plate I).

An exactly similar case is recorded by Mr. Croft in the 'Lancet,' 1890, vol. i, p. 701. The head of the humerus was removed from the axilla under the same circumstances, and is now mounted as a specimen in St. Thomas's Hospital Museum. The patient was a man aged 33 who had fallen through a trap-door for a distance of about eighteen feet. The symptoms were much the same as those of the case which is now being recorded.

The specimen, with Mr. Croft's permission, has been brought here so that it may be compared with mine. It will be seen that the line of fracture is almost identical. *May 16th*, 1893.

2. *Specimens of hammer-toes.*

By WARRINGTON HAWARD.

THE specimens exhibited are six second toes from four persons. The toes are all well-marked examples of the deformity usually called "hammer-toe," and were all amputated at the request of the possessors on account of the pain and lameness to which their condition gave rise.

The sex and age of the persons from whom the toes were removed were as follows:

A lady, aged forty-six, both second toes were affected. A man, aged twenty, both second toes were affected. A girl, aged fourteen, the right second toe was affected. A youth, aged fifteen, the right second toe was affected.

Of the last case, John W—, æt. 15, from whom the second toe of the right foot was removed, the following family history was obtained:

(A.) *Paternal grandmother* had hammer-toe, viz. second toe of right foot.

(A's son, B.) *Father* had same deformity, viz. second toe of right foot.

B's children.—(1) Son, hammer-toe, second toe right foot. (2) daughter, hammer-toe, second toes of both feet. (3) Daughter, no deformity. (4) Daughter, no deformity. (5) Son, hammer-toe, second toe left foot. (6) Daughter, no deformity. (7) Son (this patient), hammer-toe, second toe right foot.

These toes have all been amputated at the metatarso-phalangeal joint, and show the same abnormal conditions. But the specimens have been prepared in various ways, the better to display the changes in the individual parts and structures concerned. It will be seen that the first phalanx is in a position of extreme extension, that the second phalanx is flexed on the first at nearly a right angle, and that the third phalanx is extended on the second. This position throws undue weight upon the tip of the toe, whereby the nail is thrust upwards and the skin becomes greatly hypertrophied.

The projection above the level of the adjacent toes of the first

inter-phalangeal joint leads to pressure by the boot upon its dorsal surface, to hypertrophy of the skin, and the formation of a corn with a subjacent bursa. It is the inflammation and ulceration of this corn and bursa which give rise to the lameness and pain accompanying the deformity.

In every case the extensor tendon was seen before amputation to be rigidly contracted and standing out prominently under the skin over the metatarsal bone and base of the phalanx. The flexor tendons on the contrary were found quite uncontracted and simply following the arch of flexion, as is seen in the specimens.

The lateral ligaments are shortened.

The plantar ligaments show little or no change.

The articular surfaces of the first interphalangeal joint are somewhat altered in shape by the pressure of flexion.

The specimens throw no light upon the cause of this curious deformity, but the examination of cases in an early stage shows that the hyper-extension or retraction of the proximal phalanx is the first stage in its production. *May 2nd*, 1893.

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3. *Osteo-arthritis of the joint between the first and second pieces of the sternum.* (*Card specimen.*)

By H. D. Rolleston, M.D.

The sternum shows a prominent mass of bone resembling callus, uniting the gladiolus and the manubrium sterni on their anterior aspect. On the posterior surface there is no bone between the two pieces, which are quite separate. There is no displacement of the bones to suggest that there had been any former dislocation, while there are osteophytic growths around the articular facets for the clavicles, which make it probable that the bony mass between the first two pieces of the sternum is due to osteo-arthritis. It is quite probable that this osteo-arthritis was traumatic in origin, but there is no evidence to support this view.

If this ankylosis be due to osteo-arthritis it will be a second exception to the rule that osteo-arthritis does not produce true bony ankylosis, the spine being the other.

From a man aged 65, who died from fracture of the skull. At the *post-mortem* it was thought that the sternum had been previously fractured, and that union with displacement had resulted, and so the joints were not examined for osteo-arthritis. It was only on maceration that it was shown that there had been no fracture. *November* 15*th*, 1892.

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4. *Congenital hypertrophy of the distal half of one foot and of the three middle toes.* (*Card specimen.*)

By W. G. Spencer.

The anterior half of the foot was removed by amputation at the mid-tarsal joint. The proximal part, showing the stump healed, was obtained after death, which happened from diarrhœa.

The cast of the foot was taken by Mr. Fallon.

The three middle toes are more than double in size and length as compared with the first and fifth. They were, during life, of a dusky-red colour. Beneath the metatarsals there is a thick pad of fat which was very vascular during life.

The child was seen soon after birth, and kept under observation for six months; meanwhile the affected toes appeared to be still growing at a greater rate than those of the other side.

The mother stated that when she was about three months pregnant a female lodger unexpectedly showed her a bad foot. She refused to look at the foot again, but the incident remained impressed on her mind. *May* 16*th*, 1893.

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5. *Dermoid cyst with infundibular pedicle, from a case of masked spina bifida.* (*Card specimen.*)

By W. G. Spencer.

A dermoid cyst about 1 inch in diameter, which had been cut in two, was shown. On its under surface was a tubular process having a smooth lining and continuous with the cavity of the cyst.

On the skin covering the cyst was some fine hair, and in the centre of it was an opening from which pus was discharged.

It was removed from a healthy boy aged 9½ years, the cyst being situated over the position of the spinous process of the fourth lumbar vertebra, and connected with the spine by a palpable band.

The mother stated that the swelling had existed since birth, and that it was of the size of a nut during infancy. At four years of age it swelled and discharged, and this had recurred several times.

May 16th, 1893.

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6. *Ivory exostosis of the skull.* (*Card specimen.*)

By S. W. Wheaton, M.D.

The patient, from whom the tumour was removed by an operation, was a female aged 60, who had received several severe injuries to the head. The tumour was first noticed ten years ago, at which time she began to complain of severe pain in the head on the same side as the tumour, which was situated on the right side of the head, so that its centre was just one and a half inches behind the external auditory meatus, and on a level with the top of the pinna. The pain, which had been continuous, had become more intense, and lately there had been marked tenderness over the tumour and on the scalp around it. During the last three years there had been pain shooting down the right arm and leg and marked weakness of the right arm and hand, without any affection of speech. There was also slowly developing loss of vision in the right eye, which had become complete during the last few months.

On ophthalmoscopic examination the right optic disc was found to be in a condition of atrophy, and surrounded by large white patches of choroidal atrophy. The fundus of the left eye was normal.

It was thought probable that there was a corresponding growth on the inner aspect of the skull, exerting pressure on the medulla in the direction of the foramen magnum.

The tumour was removed, together with the portion of bone from which it was growing, by trephining. The dura mater beneath

the tumour was very vascular and firmly adherent, but there were no signs of any extension of the growth inwards. The dura mater was not punctured. The headache and other symptoms were not relieved at all by the operation, and the patient died nine days later from bronchitis.

At the autopsy no growth was found in the interior of the skull, but old pachymeningitis was present all over the right hemisphere of the brain, which was firmly adherent to the dura mater. The dura mater itself was greatly thickened, especially at the base of the skull and in the posterior fossa, and its inner surface was covered with a thick, firm layer of lymph. The right optic nerve was markedly wasted as far as the level of the chiasma, but no wasting could be traced above this level. There were no other signs of disease in the body except general acute bronchitis affecting both lungs. The tumour consists of a cone-shaped process of ivory-like hardness, which on section is seen to be covered with an outer shell of softer bone, which is continuous with the outer table of the skull. The apex of the tumour is a little more than half an inch above the level of the surrounding bone, and its base is one inch in diameter. At the apex is a small secondary bud-like process.

It is most probable that the pachymeningitis and the exostosis were both caused by the original injury to the head.

December 20th, 1892.

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7. *Soft fibroma from the synovial bursa of the little finger.*

By Samuel G. Shattock.

An elongated, cylindrical, and lobulated tumour, five centimetres in length and two in greatest breadth. It was removed by Mr. Clutton from a woman aged 59, a nurse, who had noticed a swelling in the right palm for nineteen years, and lay beneath the palmar fascia, reaching from the distal aspect of the pisiform bone to the base of the little finger. It is smoothly covered with a thin capsule of connective tissue, and presents in section distinct indications of a lobular construction.

Histology.—The tumour, which must be classed as a soft fibroma,

consists of ill-defined lobes united by a looser connective tissue, not very sharply differentiated, however, from the former. The lobes themselves are composed of finely fibrillar ground substance, abundantly traversed by elongated and closely packed cell-nuclei. The tissue, as discernible by the arrangement of the cell-nuclei and direction of the fibrillar substance, is disposed in intersecting fasciculi. There are nowhere any giant-cells.

Remarks.—In the thirty-eighth volume of the Society's 'Transactions,' p. 324, a very extensive growth connected with the anterior carpal bursa and its extension to the index finger was recorded by Mr. Sydney Jones. The tumour formed a large swelling in the palm, which extended from above the anterior annular ligament to the base of the second phalanx of the index finger. Of this specimen I made an examination at the time, and, like the present, it is a soft fibroma.

Similar fibromata are met with also in other forms of synovial bursæ, of which a good example is described in the same volume by Mr. Makins.

It is in connection with tendon sheaths that the occurrence of giant-celled sarcoma has been many times observed. Mr. Makins (loc. cit.) adverts to one such that came under his notice, and gives references to similar cases by Nélaton, Wordsworth, Broca, Guérin, Czerny, and De Pezze.

Since then Heurtaux ('Archives générales de Médecine,' Feb., 1891, cited in the 'British Medical Journal,' March 14th, 1891) has recorded other instances. He observes that such growths spring from the sheath of the tendon, which is completely involved with its synovial membrane; the other tissues are merely displaced. "When examined with the microscope some bands of fibrous tissue —the remains of the sheath—are still visible, and the intervals are filled up with embryonic cells, round and spindle-shaped, as well as myeloplaques." The tumour is "upon the palmar surface of the metacarpo-phalangeal joint or of a phalanx, of the size of a hazel-nut, or even a walnut." "There is no pain in it, and its course is slow."

In regard to this subject, it is important to observe that under the common term tendon-sheath two distinct structures are sometimes included, viz. the dense fibrous sheaths confining, say, the flexor tendons to the phalanges, and the synovial bursæ lying in the palm, with which the first are not co-extensive.

The simple tumours arising in synovial bursæ are fibromata, and of this nature are most of the non-infective new growths of synovial membranes generally.

The myeloid sarcoma, on the other hand, is essentially a tumour of the fibrous sheath of the tendon, and the presence of giant-cells in such tumours is not a little interesting. It is difficult not to associate their presence with the continuity of the tendon-sheath and bone, and to regard these cells as arising in histological residues left during development, the complete differentiation of the structures mentioned not having been properly accomplished.

February 7th, 1893.

VI. DISEASES, ETC., OF THE DUCTLESS GLANDS.

Adrenal disease in infants.

By W. L. Wainwright, introduced by S. G. Shattock.

E. W—, female, aged 2 months, came under treatment on October 8th, 1892, suffering from purulent ophthalmia. The child was pallid and emaciated, but showed no sign of actual disease except in the eyes and lungs, where there was some broncho-pneumonia. There was no pigmentation, and though sickness was frequent it was no more so than is common in sickly infants.

Slight improvement took place under careful nursing until October 28th, when three convulsive attacks occurred; both arms became suddenly swollen, congested, and almost black, and early the next morning the child died in a state of collapse.

At the *post-mortem* examination patches of consolidation were found in both lungs, and an empyema containing one or two drachms of very fœtid pus at the left base. No sign of tubercle was found, and all the other organs seemed healthy except the adrenal bodies. These are shown to night, one is entire, of the other, part was unfortunately lost. The entire organ measures $1\frac{1}{8}$ inch in length by $\frac{5}{8}$ inch in breadth, and $\frac{1}{4}$ inch in thickness; it is tough, pale, and slightly nodular in appearance. On section the organ presents a cortical layer of apparently normal tissue, bounded internally by a band of brownish pigment, internal to which is a partly translucent, caseous-looking mass which felt gritty under the knife.

Under the microscope the outer layer is seen to consist of the normal columns of cells, with here and there patches of broken-down tissue. The band of pigment is evidently the remains of old hæmorrhage, and the central area is seen to be made up of a coarse fibrous network, containing in parts small cells, but for the most

part enclosing mulberry-like calcareous masses and a few patches of dark pigment. No tubercles are seen, and there is no infiltration with leucocytes or anything to suggest recent or present inflammation. The question arises, from what disease have these organs suffered?

In looking through Greenhow's 'Monograph' and the Report of the Committee of the Pathological Society on the ductless glands, out of 333 cases of disease of the adrenal bodies recorded I have found twelve cases in which the following diseases were present unaccompanied by symptoms of Addison's disease, viz. cancer, sarcoma, hydatids, amyloid disease, cystic and fatty degenerations, and apoplexy. There are also eighteen cases of tubercular disease without symptoms of Addison's disease. Syphilis is not mentioned as having been present in any case, or as being even suspected as the cause of the conditions found. The only diseases which need concern us here are tubercles, syphilis, and apoplexy.

Taking tubercle first, as far as the mother could tell there was no history of tubercular affections in her family, and of the father we known nothing as the child was illegitimate. The age of the child raises another point; if this is a tubercular affection how are we to reconcile it with the generally accepted statement that tubercular manifestations do not occur *in utero*, for in this case the disease must surely have been of more than two months' duration. The earliest recorded case of Addison's disease is one by Mr. Jonathan Hutchinson, in a boy aged eleven years.

Internal evidence does not carry us much further, for the description given by Greenhow of microscopic sections corresponds in a great degree with the specimen shown to-night, for though he says that in early cases there was found a general infiltration with inflammatory corpuscles, in later cases no such condition was found, but only a caseous or calcareous mass, in some cases showing even no cells present at all. Tubercles, Greenhow states, have only very occasionally been found, and yet he assumes these latter to be of tubercular origin; if they are to be accepted as such, this case has equal claims to that origin. Dr. Alexander Bruce reports a case in the 'Path. Soc. Trans. Lond.,' vol. xvii, in which there were no symptoms of Addison's disease and in which, with like appearances, neither he nor Dr. Wilson Fox were able to detect any direct evidence of tubercle in this or any other organ.

Hæmorrhages are said to be rare in tubercular affections of these

organs, but Gulliver, in 1888, reported in the 'Path. Soc. Trans. Lond.,' a case of undoubted tubercle in which these were present.

With regard to syphilis we have no evidence either way, though the child being the illegitimate offspring of a prostitute makes the presence of that disease not unlikely. Lastly, with regard to simple "apoplexy," so called. Greenhow reports three cases in adults, one of whom died from pneumonia, one from cerebral congestion, and one from phthisis. Mattei of Sienna reported a case in a man aged sixty, who died complaining of severe abdominal pain, and in whom no other organ was found to be diseased. He also reports the same condition in a child who died at birth, owing to pressure on the cord.

In the 'Lancet,' vol. ii, 1857, a case is reported of a medical man who died after a prolonged illness, attended by severe and continual abdominal pain; *post mortem* the capsules were found to be full of clot, and no other disease was detected.

The same condition has been reported in a considerable number of cases in children still-born or dying rapidly after birth, but no special symptoms have been reported.

In the case reported to-night there had certainly been hæmorrhage, but whether this was the whole change, or only a constituent part of some former lesion, is certainly doubtful.

December 20th, 1892.

VII. DISEASES, ETC., OF THE SKIN.

1. *Ulceration of the arm following vaccination in a case of hereditary syphilis.*

By S. W. Wheaton, M.D.

The child from whom the specimen was removed was well nourished at the time of birth, but the mother said that it had "snuffles" from the first.

Owing to the mother being confined in an infirmary the child was vaccinated when seven days old, and in four places. No result followed vaccination for seven days, at which time "large white blisters" appeared on three of the points of inoculation. These blisters continued unaltered for another seven days, when they burst, discharging "matter," and leaving three deep holes in the skin.

Simultaneously with the bursting of the "blisters," similar ones appeared on the skin of the abdomen, and later on the face, arms, and legs, leaving raw places when they burst and discharged their contents. The ulcers in the situation of the vaccination marks continued to enlarge, and two of them coalesced; so that when the child was admitted into hospital, six weeks after vaccination, two circular ulcers existed, each larger than a half-crown, surrounded by a dark red areola, and having slightly irregular sharply-cut margins. The anterior ulcer was covered with a thick crust of dried blood, the posterior one having a yellow granular floor with very slight purulent discharge. The axillary glands were not enlarged, but the arm was much swollen. There was a general pemphigoid eruption. The greater part of the epidermis of the palms of the hands and soles of the feet and posterior aspect of the legs and thighs had been cast off, leaving raw, red, shining surfaces, with slightly serpiginous outline. The scrotum was unaffected. The skin of the trunk was dry and scaly and covered with

brown stains; that of the face was yellow, wrinkled, and fissured, with many circular raw surfaces and thick adherent scabs, composed of dried blood. The dark red staining could still be seen surrounding the ulcers, even after prolonged soaking in spirit.

The lips were yellow, dry, and leathery. Mercury was at once given, iodoform powdered on the ulcers, and the child placed in a boracic bath. The ulcers on the arm at once improved, and when the child died, four day later, the anterior ulcer had almost healed and the posterior one was covered with a thick scab. The mother had been pregnant eight times, with only one living child; the birth of the patient had been preceded by two miscarriages, and two premature labours in which the children did not survive. The mother also presented some suspicious copper-coloured spots on the face.

The specimen is brought forward because I believe that sufficient attention has not been drawn to the effects of vaccination in children suffering from hereditary syphilis, and because it is one of those cases in which the public may think that syphilis has been inoculated. It is clearly not an instance of inoculated syphlis, since the earliest recorded time of the appearance of the secondary eruption in these cases is nine weeks after vaccination, whereas in the present case it appeared fourteen days afterwards. A few cases have been recorded in which syphilis was inoculated by vaccination, notably those of Mr. Hutchinson's; also cases where ulceration of the arm has occurred in healthy children after vaccination, as the result of neglect, infection from erysipelas, and great uncleanliness. I have not been able to find any reported case in which the connection between the ulceration of the arm and hereditary syphilis was clearly shown, as in the present one.

The presence of hereditary syphilis is, I believe, a frequent cause of phagedænic ulceration in infants; the ulceration may follow any lesion of the skin or mucous membranes.

The present is the third instance in which I have seen it follow the lesion produced by vaccination. The two previous cases recovered, but they differed from the present one in that they were older children, and had already passed through the stage of hereditary syphilis, in which they had presented general eruptions. This form of ulceration may follow impetigo in syphilitic infants, also the lesions produced by the separation of the umbilical cord. It may also occur on the palate in connection with a mild tonsillitis

in a syphilitic infant. Whether that obscure disease, noma of the mouth and vulva, is due to the same cause is not so clear, but it is extremely probable. There is no doubt that it is impossible to avoid the vaccination of children who may appear to be perfectly healthy at the time, but who may be suffering from hereditary syphilis. The earlier the age at which the vaccination is performed the greater the risk, as is well shown in the present case. The effect of the vaccination in hurrying on the development of the symptoms of hereditary syphilis is of great interest, and was very marked in the case in point. Mr. Shirley Murphy says in his article in 'Heath's Dictionary of Surgery' that the last-mentioned effect occurs whether human or calf lymph is used; also that the syphilitic eruption generally appears on the tenth day after vaccination. This is a point of great interest, inasmuch as the general eruption of inoculated smallpox appears on this day, and also the erythematous rashes which often appear after vaccination; all these facts pointing to a profound influence acting upon the skin at this time, both in variola and vaccinia. In the present case bullæ were noticed on the skin on the fourteenth day, although it is probable that a rash was present on the tenth day, but was not noticed until the bullæ appeared. The effect of the hereditary syphilis on the course of the vaccination is also of interest, the appearance of the vesicles being postponed until the seventh day in this case and the papular stage being suppressed entirely. *December 20th*, 1892.

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2. *Bacteriological examination of the contents of the distended hair-follicles in a case of (?) contagious comedo.*

By Samuel G. Shattock.

The case, one of a series in which a mother and three children were affected, was observed by our lamented colleague Dr. W. B. Hadden, at the Children's Hospital, Great Ormond Street.[1]

[1] The mother had been suffering for about ten days before the family was seen. The children included a boy aged 7, a girl aged 5, and a boy aged 3½, affected severally six, five, and four months previously. The distribution in all was the upper part of the forehead and adjoining portion of the scalp.

After sterilising the surface, the contents of one of the comedones on the forehead were pressed out and transferred on the öse to the surface of nutrient gelatin. A pure culture was obtained, which has presented uniform characters through many generations and possesses marked vitality.

The macroscopic characters may be described from notes selected from the daily examination of a fresh subculture.

Nutrient Gelatin; Streak Culture.

November 28th, 1892.—I inoculated a tube of gelatin from a jelly tube carried on in July, 1892. Kept at an almost constant laboratory temperature, the room being heated both day and night.

December 1st.—Growth along the whole of the streak; of a whitish colour, fairly uniform in thickness, with points of thicker growth; surface finely granular. By transmitted light pale brown, the thicker points appearing darker than the rest. No liquefaction.

Microscopic examination shows, as did the examination of previous cultures, that the organism is a micrococcus; there are no chains or other special groupings.

5th.—Same report. (I carried on two broth tubes from the upper end of the growth.)

7th.—Thickness increased; no obvious increase in area; by direct light the culture is of a greyish-white colour, brown by transmitted. No liquefaction.

January 7th, 1893.—Increased in thickness and spreading at the sides. The growing border is thinner, finely crenate and radially striated. No liquefaction or sinking of the medium.

February 10th.—Nothing further to note. No growth of any kind into the substance of the jelly has taken place.

Cultures in Broth.

These were kept at 35° C., and always showed the same characters.

Turbidity soon appears, and in four or five days there has formed at the bottom of the turbid fluid a consistent white mass of growth. When shaken this presents a noteworthy but not a diagnostic appearance. If the tube be shaken in a circular way the culture rises in the form of a twisted tress, with the upper end frayed out or hairy; whilst the lower when freed from the bottom of the tube is rounded or truncate.

In time the broth becomes quite clear by subsidence, but on

shaking the tube the culture rises into the fluid and still presents the characters described.

Examination of the turbid fluid after six days shows a fair number of micrococci in twos, fours, and less regular aggregations.

Nutrient Gelatin; Stuck Culture (Laboratory temperature).

May 19th.—The subculture started from a recent broth culture.

20th.—Obvious increase in the material introduced.

24th.—Along the needle track is a line of white growth, and at the surface a thin circular extension, about ·4 cm. in diameter, the edge of which is finely crenate. The growth in the track consists of an aggregate of minute white spherical colonies.

June 1st.—The culture presents the same characters; the surface-growth is milky white and ·5 cm. in diameter.

12th.—Nothing further to note.

Glycerin Agar; Streak Culture, kept at 35° C.

May 19th.—The subculture started.

20th.—Obvious growth in a thin sheet over the track made by the öse; whitish by direct light, pale brown by transmitted. Where thinnest it consists, as seen with a lens, of minute circular colonies which subsequently coalesce to form a moist continuous film.

24th.—Full growth; milky-white by direct, brown by transmitted light; growing edge thinner than the rest, and undulating or more finely crenate.

June 1st.—No obvious extension of the growth.

12th.—Nothing new to note.

Repeated rubbing of a fresh broth culture into the skin between the shoulders, well washed before, and not again during the experiment, gave no results. Similar experiments on monkeys were equally negative.

The micrococcus corresponds, so far as I can find, with none of those yet isolated from the surface of the body, whether pathogenic or not, that have received distinctive names. At least, out of the twenty-four recounted by Sternberg ('Manual of Bacteriology,' p. 574) no such is included.

Amongst the coccus forms are given diplococcus albicans tardus, diplococcus citreus liquefaciens, diplococcus flavus liquefaciens tardus, staphylococcus viridis flavescens; and

amongst pathogenic cocci, staphylococcus pyogenes albus, staphylococcus pyogenes aureus, streptococcus pyogenes, Demme's Diplococcus.

Nor does the organism correspond with any of those named of the non-chromogenic group diagnosed by their growth taking place on nutrient jelly at the room temperature without any accompanying liquefaction.

It would appear, however, to be one of the unnamed cocci, isolated from the normal skin by Bordone-Uffreduzzi.

This observer[1] has described seven kinds of micro-organisms from the normal skin. They include the well-known *Bacillus epidermidis* and *Bacterium graveolens*, and in addition to these, five forms of micrococci.

Two of the latter are chromogenic, and may be at once dismissed from consideration.

Among the three non-chromogenic, one liquefies the gelatine very early, and may in like manner be excluded.

But another grows on gelatine in white colonies, without inducing liquefaction. This species never grew in a culture made from the epidermic scales of any part of the body, but invariably from the hair roots plucked from the head, beard, axilla, and pubes. The cocci are connected in twos, or usually in irregular masses; and grow on all media at the room temperature, 15° to 20° C.

Pure cultures yielded no results in guinea-pigs and rabbits, whether injected beneath the skin, into serous cavities, or directly into the blood; inunction was equally negative.

In the horse a contagious acne, terminating in suppuration, has been described as due to infection from the saddle. From the lesions a bacillus has been isolated, by rubbing which into the skin deprived of its epidermis the disease is reproduced.

From other cases of acne in the horse, however, only the *Staphylococcus pyogenes-aureus* has been cultivated (Cornil and Babes, 'Les Bactéries,' 1890, tome i, p. 394).

So far, then, as the evidence goes in the present case, the presence of the micrococcus in the distended hair-follicles can be regarded only as an epi-phenomenon. *May 16th*, 1893.

[1] 'Fortschritte der Medicin,' Bd. iv, 1886, p. 151.

3. *Traumatic epithelial cyst of hand.*

By Raymond Johnson, M.B., B.S.

A boy, aged 7½ years, fell in the road, cutting his left hand at the upper end of the thenar eminence. The wound healed in about a fortnight. The scar remained tender, and two and a half years later a painful swelling formed beneath it. This swelling had only been noticed for three weeks when the boy first came under my observation. There was then a small scar over the upper and inner part of the thenar eminence of the left hand. Beneath the scar was a small firm nodule, loosely connected with the skin and very tender when pressed.

A short incision was made over it, and the cyst which is exhibited was removed. The cyst was quite unattached to the skin, and shelled out with the greatest ease from its bed in the subcutaneous fat. It is oval in shape, and measures three eighths of an inch in its longest diameter. It consists of a thin wall, dense in texture and pearly white in colour, and contains a white crumbling material.

Examined microscopically there is found to be a complete absence of fibrous tissue from its wall. Most externally there is an epithelial covering consisting of several layers of small cells, and within this are laminæ of horny epidermis. The contents consist of epithelial scales and granular *débris;* no cholesterin crystals are present.

The occurrence of dermoid cysts on the fingers in positions not explicable on developmental grounds has been long recognised. There is, moreover, strong evidence to indicate their relation to antecedent injury. Thus in a case reported by Mr. Poland ('Transactions,' vol. xxxv, p. 399) as a sebaceous cyst of the finger, the patient, a boiler-maker, had run a piece of steel into his finger three years previously. In the same volume Mr. Bowlby describes a specimen from the dorsal surface of the index finger of a woman aged sixty-two, in whom frequent pricks with a sewing-needle seemed a likely cause. In Mr. Barker's case ('Transactions,' vol. xxxvii, p. 478) the cyst occurred on the palmar aspect of the proxi-

mal phalanx of the right middle finger of a young man, in whom amputation had been performed at the distal joint for injury some years previously. In his Hunterian Lectures on 'Evolution in Pathology,' Mr. Bland Sutton refers to an interesting article on the "Pathology of the Finger" by Polaillon ('Dictionnaire encyclopédique des Sciences médicales'). He states that dermoids occur more often on the palmar than the dorsal aspect. He does not quote any cases in which definite injury is mentioned, but states that all the cases were workpeople or soldiers, who attributed the cause of the tumours to the hard work they had done. Mr. Sutton describes specimens in the museum of the Royal College of Surgeons from sheep and oxen, in which punctures with the spiked sticks of the drovers or other sharp bodies are likely causes. In its occurrence on the palm of the hand I believe this specimen stands alone. A small mass of cells from the Malpighian layer of the skin must be supposed to be transplanted into the subcutaneous tissue by the injury. Here the cells continue to divide and undergo their usual epidermic transformation, with the production of a cyst containing the products of the cell degeneration. In this specimen it is remarkable that no fibrous wall was produced by the irritation of the epithelial growth. As already mentioned, the outermost layer is cellular, and this was on all sides in contact with unaltered subcutaneous fat. For this reason it would seem that the name 'epithelial cyst' is more accurately applied to this specimen than that of 'dermoid.' *November 1st*, 1892.

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4. *Rodent ulcer of the male breast.*

By H. Betham Robinson, M.S.

The man from whom the above was removed was aged 60, and was admitted into St. Thomas's Hospital May 21st, 1890, under the care of Mr. Anderson.

He thought he had had a little lump in the nipple region for some years. In 1887 he had noticed some serous discharge, which apparently came from the outer side of the right nipple (its origin was from the surface, and not from the ducts); this formed crusts.

There was some tenderness, but no particular pain. In December, 1889, it was discovered that he had a sore on the outer side of the nipple involving the latter; its surface seemed raw and its edges raised. The sore gradually grew, and in April it had extended beyond the outer margin of the areola, and was now painful.

On admission there was an irregular ulcerated patch, the size of a half-crown, with everted edges. The surrounding tissues were not indurated, and only the outer half of the nipple was destroyed. The axillary lymph-glands were not enlarged.

The growth was freely removed; the axilla was opened and some glands dissected out, but they proved on section quite healthy.

There was no recurrence, but he died eighteen months after operation from chronic bronchitis and emphysema, to which he had been a victim for some years.

Histologically the growth is seen to be mainly epithelial. The free surface of the ulcer shows granulation tissue with degenerated epithelium in its meshes. Passing down from the free surface are columns of cells, which unite together to form a sort of network. These epithelial columns show well-marked columnar epithelial cells at their periphery. In the interior the epithelium is more spheroidal, in no part showing any tendency to become squamous or to form epithelial nests. The connective tissue between the columns is singularly free from round-celled infiltration.

January 31*st*, 1893.

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5. *Melanotic carcinoma of skin.*

By J. HUTCHINSON, jun.

FROM the category of "cancer" a considerable number of new growths have been transferred to the sarcomata. With regard to melanotic tumours commencing in the skin, it seems that it would be correct in several instances to perform a reverse process. It is, however, certain that both pigmented sarcoma and carcinoma may originate in the skin, and which is the most frequent must be left for further observation to decide.

The following case is an undoubted example of true carcinoma,

and at the same time it explains the difficulty which may be met with in other cases in deciding to which group the pigmented tumour should belong.

On the thigh of a woman aged 40 (a patient of Dr. Cory's), a growth, warty and quite black, developed. It projected considerably above the general skin surface, and measured nearly two inches in longest diameter. It was recognised as being melanotic, and was excised January, 1891. Recurrence within a few months of a small black nodule took place close to the scar [this was illustrated by a coloured drawing].

The specimen shows the new growth to have a firm, coarsely lobulated surface, of a dark but not uniformly black colour. On vertical section the skin alone seems to be affected, the subcutaneous fat being normal.

Microscopical examination.—At the margin of the tumour the transition of the epithelium into the pigmented part is readily made out, there being both down- and out-growth. Lobules, closely packed with as little intervening stroma as possible, are made up of masses of cells of a distinctly epithelial nature, some pigmented and others not. Cell-nests are found in moderate abundance, and here and there the central cells of these nests are deep brown in colour. But the most interesting point to notice is that the cells of the new growth are, although epithelial, of very small size and deficient in distinctness of outline. They resemble in these respects those found in the down-growths of rodent ulcer, and, as in the latter, are developed from the rete mucosum of the skin. In fact, if a specimen were taken of the centre of the tumour alone, one would almost certainly say that it was a melanotic sarcoma with an alveolar arrangement.

The pigment is curiously grouped; in some of the lobules not a single cell will contain any, in adjacent ones the pigmentation is uniform. Another curious feature is that scattered cells, wholly pigmented, are present in the corium which do not seem to be epithelial. This fact has been noticed in cases described by other writers.

It is probable it will be found that a considerable proportion of melanotic growths commencing in the skin are carcinomatous, and not sarcomatous. That there is any decided difference as regards secondary deposits in the lymphatic glands and general malignancy is not likely. It is remarkable what difference obtains in

these respects between one case of melanosis and another. The melanotic growths commencing in the choroid as a rule bring about the death of the patient by wide-spread secondary deposits within a few months; although several cases are on record in which, after prompt removal of the eye, no recurrence has taken place.

Whilst working at Moorfields Hospital I had the opportunity of observing a case of large melanotic tumour of one eye, which had perforated the sclerotic and invaded the tissues at the back of the orbit. Mr. Waren Tay excised the eye, but found it impossible to remove all the growth. Five years later the patient again presented herself. There was a frequent discharge of coal-black matter from between the lids, but there was no evidence of any secondary deposit, and the patient was in very fair health.

VIII. MORBID GROWTHS.

1. *Fatty tumour from the lip.*

By Walter Edmunds, M.D.

The little tumour was situated on the inner and left side of the lower lip. It was nearly spherical and about an inch and a quarter in diameter. It lay immediately under the mucous membrane, which was stretched over it but not ulcerated.

On microscopic examination the tumour proved to be a lipoma.

Adenomata within the lip are well known, but I have found no mention of fatty tumour in this situation.

The specimen is preserved in St. Thomas's Hospital Museum.

December 6th, 1892.

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2. *A large specimen of lamellar or corneal fibroma.*

By Samuel G. Shattock.

[With Plate II.]

The tumour was found unexpectedly at the autopsy of a man who died a few days after admission into St. Thomas's Hospital, of causes quite unconnected with it.

It lay between the bladder and rectum, in the subperitoneal tissue, and loosely connected with the outer surface of the latter, so loosely that it might readily have been torn from it.

The new formation, which is regularly spherical in form, and

has a smooth polished surface, measures two and a half inches in diameter. It is of firm consistence, and except for a very fine concentric striation, would be homogeneous to the eye.

After bisection, my attention was arrested by a small foreign body in its centre; this was a slightly curved, eroded piece of steel or iron, the small cavity in which it lay being stained with orange-coloured rust. The most central part of the formation is calcified; the calcareous centre is quite spherical and about a quarter of an inch in diameter; and in this the chief part of the foreign body lay, though a portion was embedded in the uncalcified substance beyond.

Histology.—The tumour consists of lamellæ of bright, stiff, fibrous-looking tissue. These are of fairly uniform thickness, and run accurately parallel, or rather concentrically, but not without frequent interconnections, and they are themselves finely fibrillated with the same regular parallelism, though the degree of fibrillation varies.

For the most part the tissue is devoid of recognisable cells, but in certain areas such are present in moderate numbers, without being closely aggregated. In vertical sections of the formation they appear either as deeply and uniformly stained leucocytes lying in the clefts between the lamellæ, or as cells with granular oval nuclei like those of common connective tissue; the cell body in such views appears as a mere line,—the optical section of what, as seen where the clefts are widened out, is a flattened element of considerable extent.

There are nowhere any traces of blood-pigment (Plate II, fig. 1).

Remarks.—Anatomically this formation corresponds with such as have received from Rindfleisch the name of corneal, and from Cornil and Ranvier that of lamellar, fibroma. Rindfleisch under this term includes the well-known thickenings of the splenic and hepatic capsules, and those less generally known of the pleura and tunica vaginalis. I have seen similar formations in the outer surface of large ovarian cysts.

A lamellar disposition of connective tissue obtains amongst normal structures, of course, in the cornea, but it is well pronounced also in the nerves, not only in the perineural sheath of the funiculi, but in such sensory end organs as Pacinian bodies and the structures allied to them. In man, Professor Horsley has described laminated end organs in the nerves themselves as terminal to proper nervi

DESCRIPTION OF PLATE II,

Illustrating Mr. Shattock's communication on a specimen of Lamellar Fibroma. (Page 151.)

From drawings by the author.

FIG. 1.—A vertical section of the new formation described. It consists of parallel inter-connected lamellæ of fibrillated substance, in which chemical analysis shows an entire absence of collagen. In some of the clefts (in certain parts) lie flattened corpuscles and leucocytes. (Hartnack, obj. 8, oc. 4, tube out.)

FIG. 2.—A horizontal section of the posterior tibial nerve (human) showing the laminated end organ of one of the nervi nervorum; it lies to the inner side of the perineural sheath of a peripheral funiculus, and in direct contiguity with the nerve-fibres.

The drawing is made partly on the basis of a photograph by Dr. E. C. Bousfield, and is modified only so as to correct some artificial dislocation of the nerve structures on one side of the end organ.

Fig 1

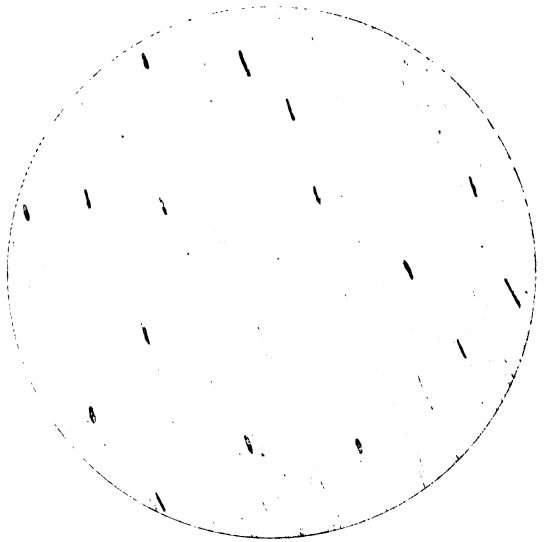

Fig. 2.

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nervorum ('Proceedings of the Med.-Chir. Soc.,' 1884, p. 196). The latter nerves he distinguishes from the sympathetic supply of the nerve-vessels (the nervi vasorum nervorum of Sappey) by their being medullated. The proper nerves of the nerve-tissue (nervi nervorum) are small medullated fibres running in the epineural sheath of the trunks, and terminating in end bulbs or Pacinian capsules.

The fibres arise singly, or in twos or threes, either from the primitive bundles (fasciculi) or their branches, and at right angles to them. Soon after leaving the fasciculus the single fibre turns and runs parallel with it, terminating usually in an end bulb which lies on the perineurium or proper sheath of the fasciculus. The end bulbs present the ordinary nucleated capsule containing a twisted core, the centre of which is perforated by the axis-cylinder.

In making horizontal sections of a human posterior tibial nerve I had the good fortune to obtain an excellent confirmation of these observations.

The end organ, which is viewed in accurate transverse section, is at once obvious (Plate II, fig. 2). It lies not on the perineurium, as was the case in Professor Horsley's observations, but altogether on its inner side in the periphery of the funiculus, and immediately contiguous with the nerve-fibres and endoneurial fibrils. It consists peripherally of four or five comparatively coarse layers, limited by flattened nucleated cells which form fine linear partitions; the laminæ themselves are quite uncoloured and transparent, but show under higher powers an extremely delicate fibrillation, the fibrils coursing horizontally and with intersections, so as to construct a fine network. To the inner side of this series of laminæ is a zone of much finer lamellæ, as in an ordinary Pacinian corpuscle, and pertaining to these are a moderate number of nuclei disposed concentrically amongst them; in optical section the nuclei are oval, and exhibit a chromatic network like most of the nuclei pertaining to the cells of the coarser and more peripheral laminæ. Between certain of the series of these finer lamellæ there are spaces which disturb their symmetry, and are possibly due to shrinkage of the central part of the body, which has dislocated certain of the innermost layers from the rest.

The centre itself is homogeneous, but there is in this no stained point indicative of an axis-cylinder. Immediately within the finely

laminated portion of the core, however, are three or four oval (?) nuclei more deeply and uniformly stained than most of the others; but in these characters they are not to be differentiated from certain of the nuclei which lie between the finer lamellæ and others in the endoneurium, even where the latter are not viewed in horizontal section.

It is possible, notwithstanding, that these elements lying in the periphery of the homogeneous centre are moniliform enlargements of a convoluted axis-cylinder, though it must be confessed that on penetrating the section no continuity is traceable between any of them.

Whilst reflecting on this subject it occurred to me that these observations might furnish an explanation of the multiple tumours which occur along the nerves, and are associated in certain cases with fibromata of the skin (fibroma molluscum).

With the object of testing this hypothesis I carefully examined three typical examples of such where the nerve-trunks bore large numbers of minute fusiform or oval tumours, so thickly that the nerves, when unravelled, much resembled strings of beads. The result, however, was disappointing, for of all the tumours examined there was none that could for a moment be classed as a lamellar fibroma suggestive of an origin in such stratified bodies as the end bulbs of nervi nervorum or Pacinian corpuscles.

The cases selected for examination were Nos. 3912 (sciatic), 3919 (sciatic), in the College of Surgeons' Museum, and that described by Dr. Payne in the thirty-eighth volume of the Society's 'Transactions' (St. Thomas's Hospital Museum, No. 1923, Brachial plexus).

I chose only the smallest tumours, ones ranging from 1 mm. to 2 mm. in cross-sectional diameter.

It will be sufficient to say that they were all soft fibromata of the common fascicular type, composed of bundles of fibres intersecting almost up to the very periphery; at the periphery itself the arrangement of the fibres tended to be concentric, as in all such formations, but to no peculiar degree.

Virchow cites two cases, however (Tumours; neuroma), in which fibromata, in connection with nerves, exhibited a distinctive laminar construction.

One was a case where Marx (Dupuytren, 'Leçons orales,' Brux., 1836, p. 274) had removed a tumour the size of a pea from the

subcutaneous tissue of the inner side of the knee; in this there were found concentric layers as in an intervertebral disc.

In the second case, Axmann ('Beiträge zur mikroskopischen Anatomie u. Physiologie des Ganglion-Nerven-systems des Menschen und der Wirbelthiere,' Berlin, 1853, p. 61) removed a tumour the size of a pea from the outer side of a woman's elbow, which he regarded as a Pacinian corpuscle pathologically enlarged. According to his description it was composed of fine lamellæ, through which a nerve-fibre passed to disappear in the second half of the corpuscle; the nerve was accompanied by two blood-vessels, fine ramifications of which penetrated the growth. On this observation Virchow remarks that, however interesting it may be, it shows only that a Pacinian corpuscle may be the starting-point of a pathological formation.

In connection with this subject it may be noticed that the distribution of Pacinian corpuscles is by no means confined to the digital nerves of the hand and foot; the corpuscles are found on the pudic nerves in the penis, clitoris, bulb of the urethra and other parts, on the intercostal nerves, sacral plexus, cutaneous nerves of the upper arm and neck, the nerves of the nipple and mammary gland, on the infra-orbital nerve, the nerves to tendons and ligaments, and more rarely on intra-muscular nerves, on the periosteal nerves, and in considerable numbers on the nerves of the joints ('Quain's Anatomy,' edit. 10, vol. i, part 2, p. 342).

As to the ætiology of this new formation, the initial cause was evidently the foreign body, which presumably passed through the wall of the rectum into the subperitoneal tissue without conveying infection from the bowel. The comparative innocuousness of metallic substances as foreign bodies is well known, and a multitude of illustrative examples might be cited both from amongst surgical accidents and such scientific procedures as acupressure and the like; the little risk of infection depending on the fact that anything infective can only lie on the exterior, and what there is even here is commonly in so small amount that the living tissue-cells may readily dispose of it.

Something more, however, is necessary to account for the continued production of substance in a formation of such size. And I submit that this new factor is to be found in its position, which the functions of the bladder and rectum make one of incessant movement. In support of this I may adduce the case of hydatid

cysts in the same situation, which arise in a somewhat similar manner, from the perforation of the intestinal wall by the embryoes of the tænia and their subsequent location in the subperitoneal connective tissue. There is such a thick-walled cyst in the subperitoneal tissue of the recto-vesical fold in the museum of St. Thomas's Hospital (No. 2182). It is filled with collapsed hydatids, and has a wall ·8 cm. or three tenths of an inch in thickness, and both to the naked eye and in microscopic section it presents lamellar characters precisely like those already described; there is in this specimen, however, an entire absence of colourable cell elements, and though the fibrillation of the laminæ is in some places well marked, in others it is but faintly indicated.

In the same museum there is a thick, stiff-walled cyst (due probably to the perforation of the intestinal wall by the embryoes of *Tænia echinococcus*), which was successfully excised from the surface of the small intestine by Mr. Sydney Jones ('Path. Soc. Trans.,' vol. xxxvi, p. 213; No. 1134, Museum Catalogue). This cyst measures about 2½ by 2 inches, and its wall where thickest ·5 cm., or one fifth of an inch; its surface, from which the peritoneum is inseparable, has the characteristic look of the splenic capsule when the seat of lamellar thickening.

This cyst I find on microscopic section presents largely the same characters, but the cell elements, mostly leucocytes, are present in considerable numbers. As contrasted with hydatid cysts in such relations, those in the liver, spleen, or kidney, where movement is out of question, have not particularly thick walls.

Whether such a new formation as that described should rightly be named a tumour might be made the subject of a long discussion, but one that would serve no very useful purpose.

Of all the subjects in pathology, the ætiology of tumours is amongst the least known; and in our ignorance of this, any definition can be constructed only upon an anatomical basis.

In the present case the cause would appear to be largely mechanical; the formation is one arising from mechanical causes. But this should not exclude it from being classed amongst new growths; in the anatomical, or Virchovian sense it is one, though in its ætiology it assuredly differs as much from an ordinary fibroma as either would from a sarcoma.

Here, however, I may take up the question whether the formation under discussion is a true fibroma. The resemblance between

fibrin coagulum, when this has a lamellar disposition, and fibrous tissue is one much closer than often supposed.

In examining sections of a much thickened bursa patellæ, the difficulty of distinguishing between true fibrous tissue and its counterfeit forcibly obtruded itself into notice; for whilst the main inner portion was obviously fissured coagulum, devoid of cells and without any regular disposition, and the outermost was obviously fibrous tissue, I could not determine where the boundary line between the two lay, so closely were they alike when both were regularly laminar in disposition and cells were present in the coagulum. Whilst reflecting upon the difficulty presented the expedient of chemical analysis suggested itself.

True fibrous tissue would consist of collagen and yield gelatin, a fibrinous coagulum would yield none. This, however, does not settle the question at issue, for a tissue once fibrous, might fail to yield gelatin by reason of secondary change, as I believe to be here the case.

On submitting the problem to Dr. Halliburton he at once undertook the chemical examination, which he carried out with his well-known care, and kindly furnished the following report:

"Lamellar fibroma received from Mr. S. G. Shattock June 12th, 1893. It had been in alcohol several weeks, but this was got rid of by water, and the treatment with alcohol would not interfere with the special point I was asked to investigate, viz. whether it consisted of collagen, the substance of which white fibrous tissue is composed. The following are the experiments I performed:

"1. 0·28 grm. of the material was dried in a weighed crucible at 110° C. to constant weight. The weight of the dried residue was 0·055 grm. The material, therefore, contained 20·5 per cent. of solids.

"2. 5·25 grms. were finely divided and boiled for two hours with 100 c.c. of water. The extract was concentrated to 5 c.c. It did not gelatinise, and gave the tests for ordinary albumoses. True peptone was absent.

"3. 4·7 grms. were finely divided and heated in a sealed tube for one hour at 120° C. with 50 c.c. of water. The extract did not gelatinise after concentration and cooling, but gave the tests for albumoses. It contained also a small quantity of true peptone.

"4. A small fragment warmed to 40° C. with 0·2 per cent. hydrochloric acid for twenty-four hours became slightly swollen.

"5. 3·56 grms. subjected to artificial gastric digestion for three days nearly all dissolved. The filtrate gave the tests for ordinary albumoses and peptone; the colour reactions (*e.g.* the Adamkiewicz reaction) for gelatine peptone are different, but this substance gave the reactions of the substances obtained from an ordinary proteid such as fibrin.

"My conclusion is, therefore, that the material given me yielded no gelatine, and therefore did not consist of collagen. It consisted of some proteid difficult of solution, presumably fibrin or a body allied to it."

Although this result was not absolutely unexpected, it was nevertheless a surprise. I handed over, therefore, to Dr. Halliburton a portion of the thick laminated cyst-wall, enclosing the hydatids between the bladder and the rectum, referred to in the first part of this communication.

Macroscopically this had precisely the same characters, and was very nearly the same in microscopic section, except that no cells were recognisable in the interlamellar clefts.

Report on thick wall of cyst enclosing hydatids, by Dr. Halliburton.—"The specimen, which had been in spirit, reached me in water. It consisted of the wall of the cyst around the parasite, and would presumably be the situation where one would expect to find fibrous tissue. The material is hard, almost cartilaginous in consistency.

"1. Boiling with water at 100° and at 120° C. in a sealed tube extracted a proteid material which gave the usual proteose reactions. The extract after concentration and cooling yielded no gelatine.

"2. Another portion of the wall was subjected to artificial gastric digestion. It yielded products which gave the colour reactions of proteose and peptone from proteid, not of those from gelatine.

"3. Another portion was boiled with dilute sulphuric acid for an hour. By this time it had dissolved. The product tested with Fehling's solution gave no reduction. This negatives the presence of mucin, and of the mucin-like substance called hyalin, of which the proper cyst-walls of hyatids are stated to be composed.

"My conclusion is, therefore, that the material investigated consists of a substance proteid in nature, probably fibrin or closely related to it."

Virchow ('Tumours,' Lecture 8), in his account of hæmatocele,

mentions a diffuse thickening of the tunica vaginalis as sometimes met with in which an evident stratification is distinguishable, and in regard to the thickened areas or sclerotic plaques seen on the tunica albuginea and parietal vaginalis, which he likens to those of the splenic capsule in chronic perisplenitis, he observes that these are formed of a very dense connective tissue of which the intercellular substance has a very compact conformation, not undulatory but stiff, and very little fibrillar.[1]

Such plaques may undergo calcification; 2300, St. Thomas's Hosp. Mus., shows the sac of a hydrocele, dried and rendered transparent with turpentine; there are many thin ill-defined patches of opaque white calcareous material in its wall. The plates of calcareous substance met with in the parietal pleura have probably a like origin (St. Thomas's Hosp. Mus., Nos. 1820, 1821).

Similar formations, observes Virchow, lead to the production of pedunculated, and these, to free bodies in the tunica vaginalis; the latter, to cite his description, are characterised by a laminated structure; their size varies from a pin's head to a musket ball. On making a section through one of the larger, it is ordinarily seen to be formed in its outer part of cartilage-like layers like the sclerotic plaques of the tunica albuginea itself; in the interior there is most frequently perfect calcification, of such an extent that if the body is the size of a cherry-stone, the calcified centre has the size of its kernel.

The conformation, in short, is precisely on a small scale that of the large formation under notice, and the thickenings of the splenic capsule and tunica vaginalis are microscopically of precisely the same kind.

In the two examples from the spleen and one from the tunica vaginalis which I have examined no corpuscles were disclosed by means of hæmatoxylin. In each I hope Dr. Halliburton will be able, on a future occasion, to determine the presence or absence of collagen.

That these plaques are not of ordinary simple fibrin-coagulum is

[1] Nos. 2295, 2302, St. Thomas's Hospital Museum, are good examples. No. 2295 is a hydrocele, of which the sac is thin-walled and smoothly polished internally; over the testicle the peritoneum is here and there raised in low dendritic lines. No. 2302 is a vertical section of a testicle showing a partial hydrocele; on the side of the sac there is an extensive plaque of thickening.

told, amongst other ways, by their not responding to Weigert's staining reaction (aniline gentian violet, iodine solution, and subsequent decolorisation in aniline and xylol).

What must be assumed, I think, is that these, like the neoplasm in question, are lamellar formations of true fibrous tissue in which a secondary hyaline change has occurred; and this change has obscured, in different degrees, not only the fibrillation of the bundles, but the few corpuscles in the tissue as well.

It is quite true that coagula themselves undergo secondary changes which bring them to resemble, histologically at any rate, hyaline substance. But the reasons for regarding the several formations in question as consisting of altered fibrous tissue are: the great regularity of their lamination; the interconnection of the lamellæ, which precisely reproduces the disposition of laminated fibrous tissue; the regular arrangement of the fibrillation where this is present; the presence of connective-tissue corpuscles in certain areas; the existence of intermediate corpusculated forms where the laminæ consist of wavy fibrous tissue, as was instanced in the cyst referred to from the surface of the small intestine.

The chemistry of hyaline degeneration is a subject for further research. The substance called "hyalin," which is obtained from the proper cyst wall of hydatids, cannot be chemically the same as the hyaline substance met with in pathology, though the same term is common to both.

Indeed, in the second of Dr. Halliburton's analyses he distinctly points out that the tissue does not give the special reactions of this mucin-like substance.

It is highly interesting, however, to find that where the hyaline substance resulting from the transformation of fibrous tissue can be examined in a pure state, and in so large a bulk as to exclude fallacy, the tissue no longer contains collagen.

April 18*th*, 1893.

3. *A case of spindle-celled sarcoma of the sterno-mastoid.*

By WILLIAM ANDERSON.

THE patient, a female infant aged 13 months, was sent to me by Dr. Achard in September last. She was then suffering from a tumour of the right sterno-mastoid of seven months' duration. The enlargement had been slow and painless, and had not affected the child's health. The mother was quite certain that no swelling in the muscle was present during the first six months after birth. There was no history of syphilis in the child or parents.

On examination the tumour was found to be about the size and shape of a large walnut, hard but elastic, moveable in the lateral direction upon the deep structures, and not adherent to skin. There was an apparent torticollis, but on closer observation it became evident that this was due to the effort of the child to remove pressure from the parts beneath the growth. The lymphatic glands were not affected.

The tumour, which was diagnosed as a spindle-celled or fibrifying sarcoma, was excised on the 8th of October, with the entire length of the affected muscle from the sternum and clavicle to the skull. It was found to replace the whole of the muscular fibres, except a portion of the cleido-occipitalis which ran over its posterior surface. It was unencapsulated, and careful dissection was required to detach it from the carotid sheath. The spinal accessory nerve traversed its substance, and was necessarily divided. The histological examination showed the growth to consist entirely of spindle-cells.

The child made a good recovery, despite an attack of erysipelas appearing on the second day after the operation, and there is at present no tendency to wry-neck.

Remarks.—This is, I believe, the first recorded example of a sarcoma of the sterno-mastoid. The only case bearing any resemblance to it is one described by Dr. Frederick Taylor in the 'Transactions' of the Society in 1874, but this appears to have been a diffuse hyperplasia of fibrous tissue in a syphilitic child, and was perhaps gummatous in origin. Similar growths, however, have been found in the muscles of the extremities, and one

involving the sartorius is mentioned by Mr. Erichsen. It recurred a year after removal, and subsequently proved fatal.

It will be interesting to watch the case, with respect both to the possibility of a recurrence and the effect the extirpation of the muscle will have upon the position and movements of the neck.

December 6th, 1892.

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4. *Myxo-sarcoma of the humerus undergoing chondrification.*

By Edgar Willett, for Howard Marsh.

The specimen shown consists of one half of a left humerus (divided longitudinally) of a lady aged 54, who had noticed a swelling for rather less than a year. There is a nodular swelling about four inches long on the anterior surface of the bone; this has a thin layer of bone forming its outer wall, but is fluctuating, and could be cut through with a knife. At first sight it looked as though the disease was limited to the centre of the shaft, and that both extremities had escaped, and it was not until the bone had been sawn through longitudinally that it was seen how extensive the disease really was. The interior of the bony shell contained a reddish jelly-like substance which, at one spot situated at about the middle of the shaft, had penetrated into the interior of the bone, and had grown upwards and downwards in the medullary cavity until it had reached a point within two inches of the upper extremity and nearly as far towards the lower. The shaft is slightly expanded, but almost uniformly so; there are, however, two or three places where the cancellous tissue is perceptibly thickened.

As to the characters of the growth, at the time of the operation after incising the tumour, a small amount of a gelatinous translucent material was removed with the end of the scalpel; some of this was subsequently hardened in Müller's fluid, cut and stained with Ehrlich's hæmatoxylin. On looking at the stained sections with the naked eye it is seen that, with the exception of one or two small patches, they are only faintly stained. Under the microscope the unstained parts consist of a number of large cells, with a fine

but distinct cell-wall and a small well-stained nucleus; the rest of the contents of the patches are unstained, and are presumably of a myxomatous character. The well-stained patches consist of a homogeneous matrix enclosing large cells, which are faintly stained and contain one or two deeply stained nucleoli (hyaline cartilage): the transition from the unstained to the stained parts is not quite abrupt, as the staining gradually fades off, giving the appearance as though the myxomatous parts were becoming cartilaginous, *i. e.* undergoing chondrification. Although myxomata occurring primarily in bone are rare, they are not unknown, and it seems as though this is an instance.

The limb was removed by Spencer's amputation at the shoulder joint, and the patient made a very good recovery.

December 6th, 1892.

Report on Mr. Edgar Willett's specimen of tumour of the humerus, by the Morbid Growths Committee.—With Mr. Willett we consider the tumour to be partly cartilaginous, partly myxomatous; but as to the relationship of the two kinds of tissue we do not think any certain decision could be arrived at, *i. e.* as to whether the first had been preceded by the second, or *vice versâ*, for it is equally possible that both these forms of tissue have arisen during the growth of the tumour from a common indifferent substructure.

The author, for descriptive purposes, divides the tissue into stained and unstained portions, and regards the unstained as myxomatous, the stained as cartilaginous.

We do not entirely agree with the description, for the unstained portions consist in part of cartilage, the matrix in such spots having failed to take the dye, in which respect cartilage is very variable. The author's description of large cells with distinct cell-wall (in the unstained parts) we regard as applying to areas which are cartilaginous. There are other unstained areas which consist of true myxomatous tissue, areas without such cells, and of one of the ordinary types, viz. a finely fibrillar matrix strewn with comparatively small cells of the common connective-tissue kind, the cell body being faintly stained, sometimes irregular in outline, at others sharp, and oval or circular; the latter cells being such as are met with in, for instance, nasal myxomata.

With respect to the stained portions the coloration is due to

staining of the matrix in the neighbourhood of the large cartilage-cells; the bodies of the latter are everywhere shrunken away from their containing capsules.

R. W. Parker.
S. G. Shattock.
Seymour J. Sharkey.

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5. *Calcareous particles in a parotid tumour.*

By H. Betham Robinson, M.S.

To Mr. Sydney Jones, by whom the growth was removed in private, I am indebted for the opportunity of bringing this case before the Society.

All history of the tumour was unfortunately lost, but its histology presents some curious features worthy of record.

The growth is of an adeno-sarcomatous nature, the masses of the latter growth being separated by a well-marked connective tissue. Distributed chiefly through the periphery of the masses and in the adjacent connective tissue are granules, of varying size and form; these have a distinct appearance of lamination, and are stained of a purplish tinge with Ehrlich's hæmatoxylin. They are formed of carbonate of calcium.

These particles have not been formed in relation with any calcareous degeneration in the tumour, but are due to deposition from the secretion of the gland, possibly coincident with some hindrance to excretion of the saliva. *January* 31*st*, 1893.

IX. MISCELLANEOUS SPECIMENS.

1. *A large blood tumour developing at the mouth of a wound in a case of hæmophilia.*

By G. H. Makins.

The specimen was obtained from a boy aged 12, the subject of a comminuted fracture of the humerus. The fracture was accompanied by considerable swelling, and on the fifth day the patient had a rigor, the temperature rising to 104° F. The swelling was then aspirated, a very small quantity of blood being drawn off. On the seventh day he was admitted into St. Thomas's Hospital, the swelling then being hot and red, and the general temperature 104°. No suspicion of the diathesis was at that time entertained, and the swelling was freely incised, fluid serum and breaking-down clot being evacuated.

The temperature subsided after the incision, and the wound progressed favorably for nine days, when clot commenced to collect at the mouth of the wound, although there was no hæmorrhage into the dressings. The temperature again rose, and the mass of pink firm clot increased to the size of an orange. On the sixteenth day after the first incision the wound was again cleared of clot, and a number of loose fragments of bone removed. Very free hæmorrhage of a parenchymatous nature followed, which had to be arrested by plugging; and in the evening of the day of operation the symptoms due to loss of blood were so alarming that it was deemed advisable to infuse three and a half pints of normal saline solution. He rallied and did well for ten days, no further hæmorrhage occurring, the wound in the vein (median basilic) selected for infusion healing readily. After this period, however,

hæmorrhage from the large wound commenced in small quantities, and soon a firm tumour again formed.

At this time we had the opportunity of seeing one of his relatives, which had before been impracticable as the boy came from an orphan school, and learned that there had been several bleeders in his family (mother's cousin, female; mother's niece; one brother, and one sister). There had, however, been no death in the family from hæmorrhage, and the patient himself had not been regarded as a bleeder by his friends, although he often suffered with slight hæmorrhages from the nose.

This knowledge precluded further operative interference, and although various styptics were administered internally the tumour gradually increased in size. There was no external hæmorrhage, but the patient steadily sank, his condition being aggravated by fever resulting from decomposition of the surface of the clot consequent on the trial of leaving it exposed to the air. At the time of death the tumour, which had been growing seventeen days, extended from the front of the shoulder, the arm lying against the chest, to mid-sternum, upwards to above the clavicle, and downwards to nearly the lower margin of the ribs (see Fig. 8). It was greyish red in colour, lobulated on the surface, very firm, and on section proved to consist of laminated clot somewhat resembling that seen within an aneurismal sac, the centre containing some fluid serum. Before death its coherence almost suggested a large sarcomatous tumour.

Microscopic examination showed that in the older parts of the tumour organisation was rapidly progressing, and well-marked fibrous tissue was developing. *January 17th*, 1893.

FIG. 8.

Blood tumour in a case of hæmophilia. From a photograph taken after death. The "tumour" is somewhat shrunken and displaced.

2. *Preparations from the organs of a case of West African "Blackwater" fever.*

By S. W. Wheaton, M.D.

The preparations were made from the organs of a fatal case of "Blackwater" fever, which were sent over from the West African coast by Mr. Rendall. The disease prevails all along the West African coast, and over a great part of tropical Africa, and from the great fatality that it causes among Europeans has attracted a great deal of attention of late.

Description of the disease.—For help in collecting a description of this disease I am chiefly indebted to Mr. Crosse, principal medical officer to the Royal Niger Company, and to my friend Dr. Harford Battersby, formerly a medical missionary in Africa, both of these gentlemen having suffered from repeated attacks of the disease themselves. The disease occurs only in those who have previously suffered from malarial fever; it is excited mainly by exposure to chill or extreme heat. The patient after a preliminary stage of shivering, numbness of the extremities, pain in the loins, and general malaise, develops slight jaundice, with fever rising to 103°, and passes porter-coloured urine. In severe cases bilious vomiting occurs, and the patient dies with symptoms of uræmia. The urine is acid, contains albumen, hæmoglobin, or methæmoglobin, and deposits a grumous precipitate: red corpuscles are absent from it. Enlargement of the liver and spleen are uncommon.

The attacks recur again and again after fresh exposure to heat or chill, some cases having ten or more attacks. Nephritis and true hæmaturia sometimes follow the attacks.

From this account it will be seen that the disease corresponds clinically with paroxysmal hæmoglobinuria as seen in this country, the bilious vomiting and high mortality being the only symptoms which are absent in the case of the latter disease.

Description of the preparations.—The preparations are made from the kidneys, liver, and spleen, and stained with logwood and orange rubin. The kidneys show in their cortical portions marked swelling of the epithelium of the secreting tubules, the cells being opaque, their outlines indistinct, and containing yellow granules.

The lumen of the secreting tubules is packed with small yellow pigment granules. The tubules in the pyramids are filled with large brown amorphous masses of pigment, which can also be seen in the form of brown masses within the cells lining them in a few places. Beneath the capsule are large collections of similar pigment. The Malpighian corpuscles are unaffected. The spleen shows red-brown points, consisting of collections of amorphous pigment, and smaller masses of the same pigment are scattered between the cells of the splenic pulp. In addition there are large circular cells with large nuclei and containing pigment, which on comparison with sections of a healthy spleen appear to be about three times the size of the larger kind of cells usually met with in the spleen. The liver shows marked cloudy swelling of the cells, and proliferation of their nuclei in places, and the presence of collections of globules of yellow pigment in almost every cell. There are no blood-corpuscles to be seen extravasated in the tissues anywhere, nor any blood-pigment in the capillaries or vessels of the organs. On examination with the micro-spectroscope the pigment in the liver and spleen gives the spectrum of ordinary hæmoglobin, that in the spleen is present in too small quantities to give a definite spectrum. Owing to the slight fatality of paroxysmal hæmoglobinuria in this country, very few records of the microscopical examination of the organs exist; but in all those which I can find recorded, the plugging of the tubules by masses of brown pigment and the presence of nephritis are mentioned. In two cases the presence of red-brown pigment in the spleen is mentioned, and in a case recorded by Hofmeier it was present in the marrow of the bones also.

It will thus be seen that the pathological appearances in "Blackwater" fever coincide with those which have been found in paroxysmal hæmoglobinuria, thus confirming the clinical resemblance between the two diseases. With regard to the causation of paroxysmal hæmoglobinuria, it is now generally acknowledged by every one that the blood-corpuscles are broken up in the blood, and that the blood-pigment is simply excreted by the cells of the renal tubules, and stored up in the cells of the liver and spleen, which are not provided with any means for its rapid excretion. The blood-corpuscles are in an abnormal condition, as the result of some previous disease, and exposure to cold, possibly also heat, acts as the exciting cause of their breaking down. This has been well shown by

Drs. Bristowe and Copeman, and other observers. The causes which predispose to the breaking down of the red corpuscles are primary syphilis, enteric fever, scarlet fever, malarial fever, hereditary syphilis, and the presence of uric acid in the blood, according to Dr. Haig; and in a case which has been reported by Dr. Haig, and which was associated with Raynaud's disease, the affection followed measles. In the case of "Blackwater" fever the predisposing cause appears to be undoubtedly malarial fever, and the exciting cause is exposure to chill or heat. The greater severity of the disease in tropical Africa is probably due to the greater intensity of the exciting causes. The development of acute nephritis is probably due to the irritating effect of hæmoglobin upon the renal cells during its excretion, and is a point of great interest, since, as has been shown to be the case in paroxysmal hæmoglobinuria, "Blackwater" fever is liable to be followed by what has been called paroxysmal albuminuria. A friend who has suffered from the disease tells me that he has passed a small quantity of albumen at intervals for some months after the attacks, and after returning to this country.

January 31*st*, 1893.

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3. *A leper arm.* (*Card specimen.*)

By D'Arcy Power, M.B., for Guy Neville Stephen, Government Medical Officer, Nicosia, Cyprus.

The right arm from a case of lepra mutilans. The hand is only connected with the wrist by a fibrous band. The process was not complete, as the patient died before the hand actually dropped off. There is a typical leprous ulcer situated over the elbow.

It is unusual, Mr. Stephen says, for whole limbs to be affected as in this case. The more usual sequence of events is for a single phalanx to become swollen, the swelling being preceded by prolonged and slow wasting of a member. An ulcer forms over the affected joint, the bone, frequently half absorbed, drops out, the ulcer heals, the fleshy parts contract, and the process is repeated in another joint. The repair is so perfect that it is often difficult to make out any traces of the destructive process of ulceration except by means of the resulting deformity. *November* 2*nd*, 1892.

4. *Inoculation experiments showing the nature of the pulmonary lesions in leprosy.*

By CHARLES SLATER, M.B., for BEAVEN RAKE, M.D.

THE specimens exhibited consisted of (1) the left lung of a leper from which material had been derived with which to inoculate a series of guinea-pigs; and (2) various organs, glands, &c., obtained from guinea-pigs which had been inoculated with material from this or similar cases of leprosy.

The object of the inoculations was to determine, if possible, whether the phthisis which so frequently attacks lepers is tubercular in nature, or whether it is really a leprotic affection of the lung and part of the general disease from which the patient suffers.

This subject has been discussed by many observers ('Trans. Path. Soc.,' 1891, p. 386; 1892, p. 185), and the pulmonary lesions are usually regarded as not due to leprosy; but the experimental evidence is very scanty. Inoculation experiments were made by Damaschino ('Arch. Med. Expr.,' March, 1890), and tuberculosis was produced in the inoculated animals.

The present experiments and specimens appear to show that the pulmonary disease in lepers is not leprosy, but tuberculosis, since (1) the results of inoculation were positive—which is not the case in inoculations with true leprosy material; (2) the lesions in the animals resembled those of ordinary inoculated tubercle.

Specimens.

1. Left lung of a leper, aged 45, who had suffered from mixed leprosy for nineteen years, and for the last two years had exhibited signs of phthisis.

The lung is full of tubercles. At the apex they are yellow, caseating, and about the size of peas, while lower down they are grey and miliary.

2. An entire guinea-pig inoculated in the nape of the neck with material from specimen 1.

There is an indolent ulcer with much thickening of the surrounding tissues at the seat of inoculation. The cervical and

axillary glands are enlarged and in some cases suppurating, and the lungs and liver show scattered yellow nodules.

3. Specimens from a second guinea-pig inoculated with material from 1. They consisted of (*a*) the ulcer at the seat of inoculation resembling that in specimen 2; (*b*) caseous and suppurating glands from axilla; (*c*) the lungs full of small translucent nodules about $\frac{1}{8}$ inch in diameter; and (*d*) the spleen with several mottled dark red and yellow nodules about $\frac{1}{3}$ inch in diameter.

Bacilli indistinguishable from *B. tuberculosis* were found in the discharge from the ulcer and in scrapings from the nodules in the lung and spleen.

4. Specimens from a guinea-pig which had been inoculated with material from the lung of a leper, aged 17, who had suffered from mixed leprosy for nine years, and died with hæmoptysis. The lungs of the patient showed cavities and tubercles, and the liver, spleen, and peritoneum also were tubercular. The organs shown from the inoculated animal were (*a*) the lungs full of small translucent nodules about $\frac{1}{8}$ inch in diameter, and (*b*) the liver with many small yellow nodules about the size of those in the lungs.

Bacilli resembling *B. tuberculosis* were found in scrapings of the nodules in the liver and lungs. *March 2nd*, 1893.

5. *On Madura disease (mycetoma) of the foot.*

By Richard T. Hewlett, M.D.

[With Plate III.]

Madura disease, or mycetoma, is a well-known affection occurring in certain districts in India, and is termed by some authors the fungus disease of India. The condition has been investigated by numerous observers,[1] and in particular by Vandyke Carter, who has published a most able monograph on the subject.[2]

[1] See authors quoted by Lewis and Cunningham in 'Physiological and Pathological Researches,' Lewis, and 'Trans. Path. Soc.,' 1864 to 1886.

[2] 'Mycetoma, or the Fungus Disease of India,' London, 1874.

The disease is a very chronic one, attacking the hand and foot, and is divided by Carter into two varieties,—(1) the black or melanoid, and (2) the white or orchroid. The first of these is characterised by the presence of irregular black granules varying in size from fine dust up to masses as large as marbles, and of a somewhat brittle, coal-like consistence. In the white or orchroid variety black particles are absent; in their stead we find small cavities, the size of peas or thereabouts, containing numbers of small yellowish granules resembling fish roe, and hence known as the roe-like particles.

In other respects the two varieties resemble each other closely, though some slight differences have been pointed out between them. Both give rise to hyperplasia and distortion, to caries of bone, and to destruction of the soft tissues. Last year I published a brief note[1] on Madura foot, based on an examination of a specimen of the white variety. Kanthack had previously brought the subject before this Society,[2] and has lately published a very full account of the disease[3] in which he maintains that the white and black varieties are the earlier and later stages respectively of one pathological process. Both Kanthack and myself came to the conclusion that Madura disease, or mycetoma, is of an actinomycotic nature—a view, however, which had been suggested and maintained by Carter[4] some years before, and which had received the support of Bristowe, Ponfinck, Baumgarten, Bassini, and Majocchi.

Recently Boyce and Surveyor[5] have come to the conclusion that the black and white varieties of mycetoma are distinct from each other, and are due to different causative agents. I have now had the opportunity of examining three specimens of Madura foot, two received from Surgeon-Major Hatch, I.M.S. (hereafter referred to as Case 1 and Case 3), and a third kindly given me by Prof. Boyce (referred to as Case 2). The following is a detailed description of these specimens.

Case 1.—This is a typical example of the white or orchroid variety of Madura disease. Tissue hyperplasia is a marked feature, and gives rise to considereble distortion of the foot; the bones are carious

[1] 'Lancet,' vol. ii, 1892, p. 18.

[2] January 23rd, 1892.

[3] 'Journ. of Path. and Bacteriol.,' October, 1892.

[4] 'Bombay Med. and Phys. Soc.,' 1886, ix (New Series), p. 86.

[5] Proc. Roy. Soc.,' 1893.

and numerous sinuses are present, communicating with the exterior by mammillated apertures. There are many small cavities containing yellow granular matter, the so-called roe-like particles. Microscopically there is considerable epithelial proliferation in some places, and the vessels are much thickened. Scattered through the tissues in the unstained condition are yellowish bodies of variable size, and more or less reniform or crescentic in shape. These are the so-called reniform or crescentic bodies; they are surrounded by a radial zone, and the whole is situated in a mass of round cells, many of which are pigmented, with a fibrous capsule at the periphery. By staining by Gram's method and counterstaining with eosin the crescentic bodies are stained a deep pink, and are apparently structureless (Plate III, fig. 1, *c*); while the round cells (Plate III, fig. 1, *a*) and radial zone (Plate III, fig. 1, *b*)

FIG. 9.

Madura foot (Case 1). *a*. Nail-shaped pigment granules from the centre of a "crescentic body." (× 800.) *b*. Stunted clubs growing from periphery of "crescentic body;" section stained with hæmatoxylin and eosin. (× 1000.)

take on a fainter pink. Within the crescentic bodies ovoid or wedge-shaped areas are present, containing masses of fine filaments stained purple (Plate III, fig. 1, *d*), which resemble the mycelial filaments met with in actinomycosis. In and about the centres of the crescentic bodies there are peculiar granules of black pigment (Plate III, fig. 1, *e*). These are almost without exception nail-shaped, having an enlarged rounded head, attached to which is a tapering tail (fig. 9, *a*). They vary somewhat in length, but average about 6 μ or 7 μ. The radial zone shows a distinct radial arrangement of elements, many of which are fan-shaped

DESCRIPTION OF PLATE III,

Illustrating Dr. R. T. Hewlett's communication on Madura Foot. (P. 172.)

From drawings by the author.

FIG. 1.—Madura foot (Case 1); a well-marked specimen of the "white" variety.
a. Cellular matrix.
b. Radial zone.
c. Crescentic body.
d. Network of fine filaments (mycelium).
e. Nail-shaped pigment granules.
Section stained by Gram's method and with eosin. (× 150.)

FIG. 2.—Madura foot (Case 2). Specimen of the "white" variety.
Section showing typical well-stained clubs in the radial zone.
Stained with Ehrlich-Biondi reagent. (× 500.)

FIG. 3.—Madura foot (Case 3). An anomalous form.
Section showing a "fungus system."
a. Fungoid body.
b. Cellular matrix.
c. Large pigmented cells (more numerous than figured).
Section stained with Ehrlich-Biondi reagent. (× 175.)

FIG. 4.—Madura foot (Case 3).
Section of "fungoid body" showing honeycomb structure (Ehrlich-Biondi reagent), × 600, and to the right, three large delicate pigmented cells (Ehrlich-Biondi reagent), × 600.

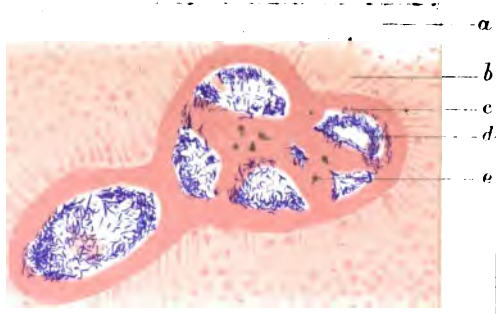

Fig 1.

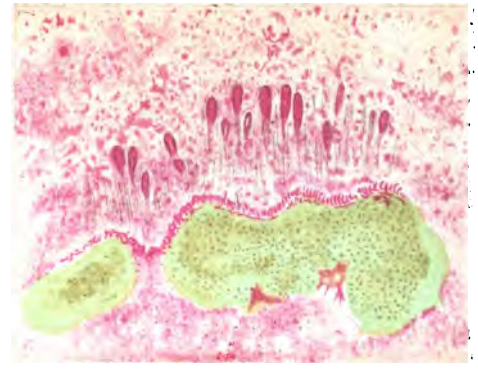

Fig 2.

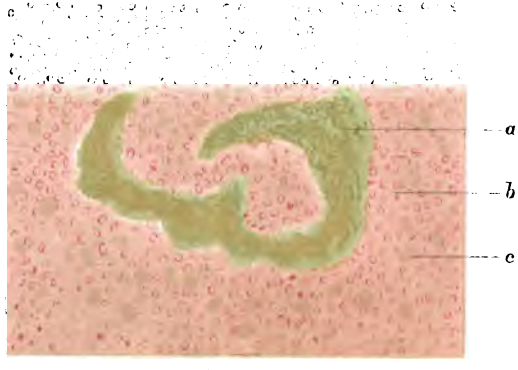

Fig 3

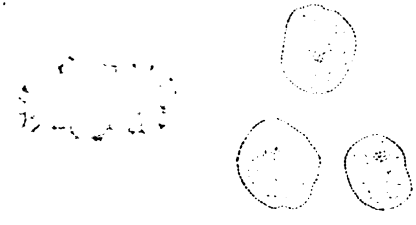

Fig 4

owing to branching. These structures are very suggestive of clubs, such as are met with in actinomycosis; they seem, however, to be in a degenerate condition, and it is impossible to demonstrate any clearly defined clubs, though there can be no doubt, I think, that they are such. By staining with hæmatoxylin and orange rubin the mycelial filaments are well shown, and in addition, situated at the margin of the crescentic bodies, small and stunted but perfectly characteristic clubs can be demonstrated (fig. 9, *b*). The same is also well seen in Case 2.

In Case 2, which also belongs to the white variety, and for which I am indebted to Prof. Boyce, much the same appearances are met with, and the crescentic bodies are well marked; the contained mycelial filaments are, however, more broken up than in Case 1. The radial zone is exceedingly clear and distinct, and I have been able to demonstrate typical giant clubs in it by staining with the Ehrlich-Biondi reagent (Plate III, fig. 2), very beautiful results being obtained with this stain. Pigment granules seem to be absent.

The microscopical appearances met with in the above two specimens show a striking resemblance to actinomycosis, and justify the statement that the white variety of Madura disease is a manifestation of actinomycosis hominis. As is usual with actinomycosis hominis (as has been pointed out by Acland and by Crookshank[1]), the clubs are not stained by Gram's method, while the mycelial elements are well shown. The opinion has been expressed that the clubs in Madura disease are gigantic as compared with those occurring in actinomycosis. This statement is only partially true, and it must be remembered that the Actinomyces is an exceedingly pleomorphic organism. The largest clubs met with in my specimens of Madura foot measured from 40 μ to 50 μ in length, and no doubt these are large as compared with most cases of actinomycosis. In a case of actinomycosis of the udder, however, examined in the Bacteriological Laboratory of King's College, the clubs were no less than 90 μ in length, and showed the same branching and fan-shaped structures which are so marked a feature in Madura disease. In another case, of actinomycosis hominis, some of the clubs were quite as long as those of Madura foot, so that "giant clubs" are by no means peculiar to mycetoma.

[1] See Reports on Actinomycosis in the 'Annual Report of the Agricultural Department for 1888.'

Case 3 is a most interesting specimen. To all appearances it is a typical example of Madura foot. There are, however, neither black particles nor yellow roe-like bodies, so that it is impossible to say to which variety of the disease it belongs.

Microscopically it differs entirely from the other two specimens. The crescentic bodies are totally absent; instead of these, and situated like them in masses of round cells (Plate III, fig. 3, *b*), are peculiar structures, which for convenience may be termed the "fungoid bodies" (Plate III, fig. 3, *a*).

A faint radial striation appears to be present here and there at the periphery of the "fungoid bodies;" but this is a spurious appearance, and close examination shows that no trace of a radial zone, such as was met with in the other two cases, exists. The "fungoid bodies" are of a hard consistence, and are irregular in shape, often, however, being somewhat crescentic. They are hardly distinguishable in the unstained condition, and cannot be stained by Gram's method. With the Ehrlich-Biondi reagent they show a reticulated or honeycomb appearance in section (Plate III, fig. 4), and much the same is brought out by staining with Löffler's methylene blue. Among the cells of the cellular matrix there are numerous large, delicate, roundish, or slightly ovoid cells, containing numbers of fine pigment granules (Plate III, fig. 3, *c*). These cells stain but faintly, are almost structureless, and their shape is fairly regular (Plate III, fig. 5).

The microscopical characters of this specimen differ entirely from those of the other two, both of which belong to the white variety of the disease, and the question arises, what is the nature of the condition in this third specimen? It is, I think, clearly not the same as in Case 1 and Case 2, and the appearances present cannot be explained by assuming that the stage of the disease is different in the two instances. It is interesting to note that Boyce and Surveyor[1] have recently come to the conclusion that the black and white varieties of Madura disease are in reality distinct from each other, and they ascribe the black variety to the action of an organism belonging in all probability to the higher fungi. Huntly[2] also describes two cases of the black variety of mycetoma in the initial stage, and in each black particles were present almost from the commencement of the disease. If this be

[1] Loc. cit.

[2] 'Glasgow Med. Journ.,' Nov., 1889, p. 344, and May, 1890, p. 339.

the case it is clear that the black variety cannot be a late stage of the white, as Kanthack has stated, for then the black particles would only be met with after the disease had existed for some time. The possibility of there being two classes of cases of the black variety has occurred to me: one class, which may be termed the "true black," distinct from the white, and in which the black particles are present from the first; and another class, "the spurious black," which is really a late stage of the white, and in which black particles are ultimately formed, produced by deposition of pigment or by pigmentary degeneration or metamorphosis. Thus it is quite conceivable that in Case 1, in which microscopic pigment granules occur, black particles might have been found later on.

The appearances I have described in Case 3 suggest that the structure I have termed the "fungoid body" is possibly of the nature of the "sclerotium" of a fungus, such as is met with in ergot; and this view would be confirmatory of the observations of Boyce and Surveyor. At the same time, from its extent, involving the whole of the foot and ankle, the disease is evidently of long standing, and yet no black particles are present. That the "fungoid bodies" are really the active agents in the production of the diseased condition there can be no doubt, for the signs of tissue reaction around them are so very marked. Hence it appears to me that perhaps we shall have to recognise a third variety of Madura disease, but in the present state of our knowledge this must be left an open question.

In conclusion it will not be out of place to draw attention to the beautiful results obtained with the Ehrlich-Biondi reagent as a stain for actinomycosis. With this staining agent the clubs can be easily demonstrated in cases where they are unstained by Gram's method, and very striking appearances are obtained.

April 16th, 1893.

Note.—Since the above was written I find that Surveyor had previously employed the Ehrlich-Biondi stain in mycetoma ('Brit. Med. Journ.,' Sept. 10th, 1892), but apparently not with great success.

6. *Observations on the histology of the yaws tubercle.*

By J. Jackson Clarke, M.B.

The yaws tubercles kindly sent to me by Dr. Beaven Rake represent three different modifications of the lesions. First, the young tubercles which have not been treated. These are prominent elevated papules as big as split peas, and present a yellow-white appearance in contrast with the surrounding normal negro skin. Clinically, Dr. Beaven Rake informs me, these tubercles have an intense yellow colour. A vertical section shows to the naked eye that the chief anatomical change resides in a great increase of thickness in the epidermis, which not only projects from the surface, but dips into the corium considerably beyond its normal level. In some of the tubercles the epidermis was a quarter of an inch in thickness. The white colour is accounted for partly by this thickening of the epidermis, and partly by the striking fact that the pigment has almost completely disappeared from the deeper layer of the rete. The second variety were tubercles which had begun to subside under treatment by oleate of mercury. These were broader than the first variety, and more distinctly papillated, but the thickening of the epidermis was very much less. One of these lesions in this stage is about as large as three sixpenny pieces placed in a row, with their edges just overlapping. Dr. Beaven Rake informs me that these give a fair idea of the naked-eye appearances of a cluster of yaws tubercles.

The third specimen is a broad and firm slice of tissue nearly as big as a florin. This is a slice of a "mother yaw." To quote from a private letter from Dr. Beaven Rake to myself: "these mother yaws are very obstinate, and often a solitary one will persist on the sole of the foot for months. After a time a new outbreak of disseminated yaw tubercles occurs, so that it would appear as if they were shut up for a time in this mother yaw."

Another form of the yaws papule is to be seen in the museum of the University College, where are preserved, Dr. Beaven Rake informs me, two hands sent by him from Trinidad showing a diffuse eruption, the "pian gratelle" of the French.

Interest in yaws has been increased by the appearance of an important monograph [1] by J. Numa Rat, of the Leeward Islands, West Indies. Here is to be found a full account of the disease, and a collection of the literature up to that date. Rat inclines to regard the disease as a modification of syphilis, which was the view of Virchow and Hebra, but which is opposed to the opinions of most modern writers. In the introduction to Rat's treatise Jonathan Hutchinson raises the question, "Is there a family of syphiloids?" Rat gives many cases in which he has observed a well-marked tertiary period in yaws, and after death has found gummata. This is contrary to the experience of Beaven Rake,[2] who has recently recorded some cases of yaws of long standing in which, by careful *post-mortem* examination, he was unable to find any trace of gummata. Beaven Rake regards the diseases as distinct. Seeing that some doubt still remains as to the relation of the disease to syphilis, I thought it would not be out of place to record the histological observations I have made on the lesions.

The chief histological records extant are those of Charlouis[3] and Pontoppidan.[4] The former regards the process essentially as a dermatitis; the latter states, "I believe that the disease has its seat in the rete Malpighii," and supports this view by recalling the fact that true ulceration rarely occurs save from caustic applications or in feeble individuals. The bacteriological observations of Beaven Rake are exceedingly timely, as very little work of the kind has as yet appeared.

It may be stated at once that all the lesions were papillomata, and that the younger ones had the same general structure as a syphilitic mucous tubercle. One of the chief features of interest was the loss of the natural pigment in the deeper cells of the affected epidermis, and the presence of peculiar ramified pigment-cells in connection with them. Neither Charlouis nor Pontoppidan mentions these cells; it may be their material was taken from whites, in whom, from the absence of normal pigment, they may not be evident. I will now pass on to consider the histology more in detail.

Examination of a young papule one third of an inch in breadth.—

[1] J. Numa Rat, Waterlow and Sons, London, 1891.

[2] W. Beaven Rake, 'British Journal of Dermatology,' 1881.

[3] Charlouis, 'Vierteljahrsh. für Dermatol. und Syph.,' 1881, vol. viii, p. 445.

[4] Pontoppidan, ibid., 1882, vol. ix.

The epithelial portion of the growth is one eighth of an inch in depth. The horny layer was greatly thickened, its layers being separated by collections of leucocytes. The prickle-cells immediately beneath the horny layer had lost their definite outlines, and presented the cloudy appearance of necrotic tissue. The chromatin of their nuclei was broken up, and the nuclei of leucocytes were intermixed with the degenerated epithelial cells. Between the latter and the most superficial loops of capillary vessels were several layers of epidermal cells which had a healthy appearance, so that there was no ulceration. In the deeper portions of the epithelium were numerous mitotic figures, all of which had a healthy appearance. The chromatin of the resting nuclei of most of the epithelial cells was collected into one or more irregular masses. This appearance is common in epithelial growths, including cancer.

The pigment of the deeper layers of the rete mucosum had entirely disappeared, and the branched connective-tissue cells were present in great numbers where the thickened epithelium met the corium.

The bodies of these cells lay either just beneath, or just within the epithelium, whilst their ramifications extended widely amongst the epithelial cells. No actual connection between these branches and the epithelial cells was made out, but from the analogy of the pigment-cells of the frog's skin, as described by Ehrmann,[1] it is possible that such a connection existed, and that the pigment had flowed from the cells of the rete Malpighii into the branched connective-tissue cells. The latter were not to be seen in the healthy skin beyond the papules, but at the marginal part of the latter they were seen among the epithelial cells, of which the pigment was sensibly diminished. The branched pigmented cells are also similar to those met with in the normal hair papillæ and the deeper part of the hair matrix, where Unna[2] describes their branches as terminating between the fibres of the hair, while Ehrmann found them entering the hair-fibres at their terminations.

The papillary layer of the corium was everywhere the seat of a dense inflammatory infiltration, which extended for a little way into the deeper part of the corium. Among the exudation-cells a

[1] Ehrmann, 'Vierteljahrsh. für Dermatol. und Syphilis,' 1885, vols. iii and iv, and 1886, vol. i.

[2] Unna, "Anat. of Skin.," 'v. Ziemssen's Cyclopædia.'

few, apparently leucocytes, presented distinct mitotic figures, and there were a few of the spindle-shaped cells containing brown pigment, such as have been described by Neumann[1] in syphilitic lesions and in normal skin.

The broader and flatter lesions, which had been treated, presented characters similar to the above, but less marked in degree. The epidermal portion was about $\frac{1}{16}$ inch in depth. There were very few epithelial mitoses as compared with those of the younger lesions. There was considerable inflammatory infiltration of the corium.

The slice of the mother yaw consisted throughout of the thickened epidermis, and contained cross-sections of elongated papillæ.

In the younger lesions pilo-sebaceous follicles and sweat-glands were not specially affected; only, where they traversed the infiltrated papillary layer, they, as Charlouis also observed, contained some wandering cells.

Sections stained with alkaline methyl blue, by Gram's or by the Ziel-Neelsen methods, showed no microphytes beyond numerous cocci and bacilli on the surface. *December 6th*, 1892.

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7. *Cultivation experiments with yaws tissue.*

Communicated by J. Jackson Clarke, M.B., for Beaven N. Rake, M.D.

A. *In ascitic fluid from a leper mixed with 5 per cent. gelatine and sterilised.*

Experiment 1.—June 21st, 1887.—Tube of this gelatine-serum inoculated with a piece of yaw tubercle from Karsi, a Hindu boy.

June 24th.—Commencing liquefaction and dirty white film on surface of gelatine.

November 12th.—Most of the nutrient material has dried up, leaving a dark yellow, viscid, treacly mass, with a penetrating dog-like smell. The microscope shows swarms of micrococci, and some short, thick bacilli. They do not take magenta well, and all colour is destroyed by nitric acid.

[1] Neumann, 'Vierteljahrsh. für Derm. und Syph.,' 1885.

A fresh tube of human serum mixed with gelatine and agar was inoculated with a small fragment of this yellow material.

18th.—No growth.

January 17th, 1888.—The surface of the serum is irregular, and there is a dirty white growth. The microscope shows cocci, some round, some long. They take magenta, but the colour is destroyed by nitric acid.

B. *In ascitic fluid from a leper mixed with* 1 *per cent. each of agar and gelatine, and sterilised.*

Exp. 2.—October 3rd, 1887.—A tube of this gelatine-agar serum was inoculated with a fragment of yaw tubercle from the arm of William Green, a negro boy, who was also suffering from tuberculated leprosy.

5th —There is a faint white growth round the fragment of yaw tubercle.

12th.—There is a thick, smooth, oily-white growth round the tubercle. In places are yellow patches, and at one side is a growth of mould.

16th.—The white smooth growth has increased. The yellow patches have also increased. The mould is stationary. The microscope shows swarms of small spores which take magenta readily, but lose their colour at once with nitric acid.

A fresh tube of gelatine-agar serum was inoculated with a small fragment of this yellow growth.

23rd.—In the groove made by the wire on the surface of the gelatine is a yellow growth. Surrounding this are flat white patches like oil paint. Outside the white patches and round the margin of the surface of the gelatine is a pale yellow growth. There is slight liquefaction of the gelatine.

30th.—The growth along the track of the wire has turned to a deep orange, and there are a few orange specks in its neighbourhood.

The microscope shows numerous shining oval spores, also a few good-sized bacilli. The rods take magenta readily, but not the spores. Nitric acid discharges all colour from the rods.

Some of the yellow growth was rubbed into scarifications on the back of a guinea-pig.

November 1st.—Scarifications scabbed over. No inflammation.

5th.—The yellow and white growths on the serum have increased, and the orange specks have turned to bright red like cayenne pepper.

16th.—The inoculated guinea-pig died. No redness or suppuration at site of inoculation. Tissues beneath quite normal. Viscera healthy. Only a few bacilli found.

A fresh tube of gelatine-agar serum was inoculated with a piece of skin of the guinea-pig taken from the site of inoculation.

24th.—There is a pale yellow growth like oil paint on the surface of the serum. No liquefaction. Microscope shows numerous cocci; they take magenta, but nitric acid destroys the colour.

A fresh tube of gelatine-agar serum was inoculated with a fragment of the yellow growth.

January 8th, 1888.—The serum has become liquid, and there is a rich salmon-coloured pellicle. Microscope shows swarms of rods and spores, which stain very deeply with magenta, but lose their colour with nitric acid.

Similar appearances were found in the tube inoculated on November 16th. *December 6th*, 1892.

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8. *"Cancer bodies" from a case of diffuse subperitoneal fibrosis.*

By R. G. Hebb, M.D.

[With Plate IV.]

The specimens were prepared from the much-thickened subperitoneal connective tissue of a male, aged 26, who was admitted into the Westminster Hospital under the care of Dr. Allchin. The patient was a coal porter, and had always lived in England. His family history was extremely good. The only previous illnesses the patient had suffered from were scarlet fever and gonorrhœa.

The present illness came on about two months before admission (October 27th, 1890) with "a cold." He soon found himself getting weak, and lost flesh rapidly. Upon the debility and emaciation soon supervened jaundice, diarrhœa, and ascites. He was tapped three times, and in all over fifteen pints of dark yellow ascitic fluid were removed. His temperature was normal throughout. He died on November 29th, 1890.

The *post-mortem* examination was made forty-eight hours after death. The body was much emaciated and jaundiced. The peritoneum and subperitoneal cellular tissue was everywhere very thick, tough, and fibrous, and the serous surface was covered with small flat elevations. The thickest parts were the omentum and mesenteries, but the parietal peritoneum was also very thick, especially about the right hypochondriac region. The stomach, spleen, and intestines were matted together and to the parts adjacent. The diffuse growth had included the parts in the portal fissure, so that the bile-duct and portal vein were compressed. The bile-duct was still patent, but its lumen was diminished. The branches of the portal vein in the liver were much distended. The liver weighed 67 oz., and was of a dark green colour. The gall-bladder, the walls of which were much thickened, contained about 1 oz. of green, very viscid, tenacious fluid. The peritoneal sac contained a large quantity of ascitic fluid, and the membrane was throughout in the condition of chronic peritonitis.

On the lesser curvature of the stomach, close to the cardiac orifice, was the cicatrix of a gastric ulcer. The intestinal mucosa seemed quite normal throughout, though the calibre of the gut was throughout much diminished and the tube much coiled. There were no enlarged lymphatics (or anything like a secondary deposit) in any part of the body. The rest of the viscera do not call for remark.

It will be observed that this growth is a fibrous thickening of the subperitoneal tissue, and that it is an example of growth by direct continuity. Microscopical examination showed that it consisted of fibrous tissue, often enclosing irregular areas, where the tissue was more cellular, and therefore less indurated. These areas consist for the most part of large cells, most of them being mononucleated, though some have more than one nucleus. These may be regarded as giant-cells, *i. e.* an ill-defined mass of protoplasm containing one or more nuclei, and being of larger bulk than the majority of the cells in their vicinity. When stained with phenol-fuchsin and phenol-iodine green (Russel's method) the plasma becomes pale pink and the nuclei blue.

Besides the nucleus of the tissue-cell, most of the larger cells contain spherical bodies with well-defined outline and granular contents. These spherical bodies are of variable size, but may be roughly divided into large and small. There appears to be some

DESCRIPTION OF PLATE IV,

Illustrating Dr. Hebb's communication on "Cancer Bodies." (Page 183.)

In all the figures the cell-plasma is stained pink, the cell-nucleus blue, while the "cancer body" is of a dark red hue.

Fig. 1.—A single body with scattered granular contents and well-defined outline.

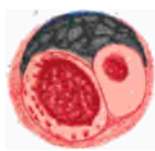
Figs. 2, 4, 5 show cells containing several small bodies having a well-defined outline and central nucleoid contents.

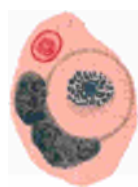
Figs. 7, 8, 9.—Cells containing one or two spheroidal bodies, differing only in size and number from those in 2, 4, 5.

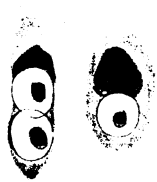
Fig. 3.—Large cell with two bodies, in one of which the contents are large and somewhat granular, in the other less granular and nucleoid in character.

Fig. 6.—Cell showing two bodies, the larger of which has stained a slaty-blue colour (meta-chromatism). In other respects it resembles the bodies in figs. 1 and 3.

Drawn under $\frac{1}{8}$ apochromatic, oc. 10, tube length 12 inches. (Powell and Lealand.)







relation between size and number. The general arrangement of these bodies is that they occupy the cell plasma, pushing the nucleus aside. The smaller ones, perfectly spherical, apparently consist of an investing membrane and central granular contents. In the larger bodies the contents may form an isolated irregular central mass, or may show some radiating bands connecting them with the limiting membrane.

But in some examples the central body has a definite shape and limits (nucleus). It is circular or ovoid, with well-marked outline, its contents being homogeneous or finely granular.

The bodies alluded to can be observed without any special method of preparation in fixation and staining, although, no doubt, the contents become more apparent after being properly stained. They are identical in appearance with the cancer parasites described and depicted by Foá, Sawtschenko, and others (see 'Centralbl. f. Bakteriol. u. Parasitenk.,' xii, 1892, Nos. 1 and 6), and are usually considered to be sporozoa.

With regard to this particular case two points may be noticed: first, that it is an inflammatory growth, malignant enough, no doubt, from its clinical aspect, but not presenting the histological appearances of carcinoma; secondly, that the very frequent presence of these bodies within the giant-cells suggests an intimate connection between them. This view is strengthened by the fact that most of these giant-cells are possessed of only one nucleus. They are overgrown cells, the size of which is due to a stimulus or irritation such as the presence of an internal parasite would supply.

The specimens were fixed in bichromate of ammonia, and afterwards hardened in alcohol. They were stained with phenol-fuchsin and phenol-iodine green. But with other staining solutions the "cancer body" is equally well seen, though the appearances are a little different. Thus with tubercle bacillus stain, phenol-fuchsin and H_2SO_4 methylen blue, the nucleus of the parasite remains quite distinct, but of a pinkish hue and marked by bluish dots or lines. With picro-nigrosin almost every cell in the section seems to contain a parasite. With osmic acid in 1 per cent. solution and developed with pyrogallic acid the appearances are very similar to, but less marked than with picro-nigrosin. With logwood and acid rubin the number of parasites seen is materially diminished.

October 18*th*, 1892.

9. *Regeneration of fibrous tissue in tendon.*

By A. H. Tubby, M.S.

The microscopical appearances of the sections shown, obtained from the tendo Achillis of rabbits at varying periods of three days to thirteen months after division, support firmly the view that in the "plasma cell" we have the immediate precursor of the permanent uniting material of severed tendons, and that leucocytes play but a secondary part.

As defining the two, I have taken those cells which are the smaller and nearly uniformly round, with occasional division of the nucleus and absence of any subsequent change of form, to be leucocytic.

On tracing these cells through sections at various dates, their gradual disappearance and replacement by the other kind of wandering cells have been a further means of distinction. The plasma cells are known by their large size, great irregularity of form, large nucleus, and granular protoplasm somewhat scanty in comparison with the size of the nucleus. They are not seldom found collected into clumps; and here the various stages of tissue formation may be traced. In normal tendon they are not easy to demonstrate, but after division they are present on the third day, and probably earlier.

The plasma cells subserve two functions; they are vasoblastic and fibroblastic. In a section three days after division they are seen to be forming the boundaries of some longitudinal vacuolations in the coagulum, and from that time can be traced till they assume an arrangement of a closely united single layer of columnar or spheroidal cells forming a vessel wall. When obliteration of the vessel occurs, the vasoblasts are giving rise to young fibrous tissue (in the thirteenth week).

There can be no doubt that the majority of the plasma cells are fibroblastic from the first, the minority however passing through a vasoblastic stage of existence. In either case it is possible to trace all the stages of tissue formation from them in experiments extending over a sufficiently prolonged period. The nucleus of the cell remains rounded for a long time, but in twelve to thirteen weeks it is fusiform, and in other instances it has entirely disappeared at

this date. Development of fibrous tissue is always best marked in the neighbourhood of the vessel. It is not till the perivascular cells have become distinctly fusiform that the vessels become blocked with round cells.

In the union of tendons, the grouping of plasma cells into islets is well marked, and follows in point of time and place the clumping together of leucocytes. Giant-cells are often seen in the clumps with several nuclei. The latter may be due to ingested leucocytes.

From my observations, it seems that in the gap between the tendon stumps the plasma cells, in addition to forming fibres, have another *rôle*. From them arise cells very similar to, if not identical with, fixed tendon cells.

The part played by fixed tendon cells is very subsidiary in regeneration. For a long time it is difficult to be sure that any change occurs in them, but as time passes, the nuclei are seen at first in the centre of the cell, and then to divide with some tailing off of the cell protoplasm. But no distinct fibres ever arise from the fixed tendon cells.

Leucocytes play but a subsidiary part, they merely form a nidus or pabulum for the development of plasma cells.

The dove-tailing of old and new tendon, as observed by Paget and Adams, is brought about as follows:—In the first place there are, after division, between the tendon bundles, the latent "schlummernde Zellen" of H. Schmidt. They are seen in groups, rounded and of small size. These give rise to larger cells which, like plasma cells, form vasoblasts and fibroblasts. In addition there is an incoming of plasma cells preceded by leucocytes from the effused new material at the site of section. Subsequently the "schlummernde Zellen" and plasma cells with their resulting vasoblasts and fibroblasts become continuous.

The disappearance of red blood-corpuscles from the coagulum between the tendon stumps is in rapid progress on the third day, and on the seventh day all trace of them is lost. It is probable that the plasma cells dispose of the red corpuscles within a given area by phagocytosis, and so form the vacuolations mentioned above which precede the formation of new vessels.

Fibrin filaments in the clot disappear by the seventh day, and have nothing whatever to do directly with the formation of young fibres. *May 2nd*, 1893.

X. SPECIAL COMMUNICATIONS.

A. ON PARASITIC MICRO-ORGANISMS IN CANCER.

1. *A critical survey of recent work bearing upon the pathology of cancer and sarcoma.*

By J. Jackson Clarke, M.B.

Within the past few years, especially the last three, the results of many important inquiries into the nature of cancer and other new growths have been published. It is my intention to classify these results, and, by comparing them, to arrive, if possible, at some definite conclusions and suggestions for guidance in further investigations and in practice.

The most important part of the work to be considered is of a pathological character, either experimental or histological. The aim of most investigations has been to prove or disprove a parasitic causation for cancer and sarcoma. Many valuable clinical observations have also been published which bear on the question but only indirectly, so that I shall omit them from this brief survey.

As long ago as 1847 Virchow[1] and others[2] figured and described cells lying surrounded by firm capsules within other cells of cancerous growths.

In 1851[3] Virchow summed up his observations on these intracellular cells thus:

"A portion of a large cell with granular contents, perhaps an

[1] 'Virch. Arch.,' vol. i.

[2] Lebert, 'Phys. Path.,' pl. xviii, fig. 8, &c.; John Hughes Bennett on 'Cancerous Growths,' p. 32.

[3] 'Virch. Arch.,' vol. iii.

altered nucleus, the dimensions of which it has, becomes homogeneous and clear, like water. This portion has at first a sharp and stout wall, which very soon by the addition of fresh layers is thickened and becomes doubly contoured in every way like a cartilage cell."

In another part of the same paper Virchow figures in the cell nests of a squamous epithelioma clusters of minute rounded bodies which had also been noticed by other observers. Virchow regarded the intra-cellular cells as the endogenous offspring of the cells containing them.

Virchow and his followers[1] regard all the constituent cells of cancer and sarcoma as lineal descendants of cells which once formed part of the body of the animal in which the tumour grew.

The accuracy of Virchow's observations has been proved again and again. The only question with regard to them is a question of interpretation.

Cohnheim's well-known hypothesis as applied to cancer is thus expressed[2] by the distinguished pathologist who so lately as 1884 passed away:—"In the tumours the redundant germinal material is inherited just as a sixth finger or a thirteenth rib; and possibly, too, the feeble resisting power of the tissues is transmitted to the offspring." At the present time this theory is challenged on all sides.

The interesting observations and theories of Klebs in Germany and Creighton in England merit careful consideration. Both have recognised the presence and the importance of certain small cells in cancer. Their conclusions will be referred to again.

Bacteriology has proved that for some of the new growths it is not true that their constituent cells are all lineal descendants of the tissue-cells of the individual in whom the growth has arisen. Such growths Virchow has included in a group, the "infective granulomata." A glance at a section of a leprosy nodule of the skin suffices to show in what numbers these parasitic and morbific fungi-cells may be present.

[1] 'Centralblatt f. Bacteriol. und Parasitenkunde,' 1892, Nos. 16—18.

[2] Cohnheim, 'Lect. on General Pathology,' New Sydenham Soc. Trans., vol. ii, p. 816.

Many pathologists have tested cancer by the bacteriological methods; in the majority of cases the results have been negative, or have been shown to be erroneous where microphytes were accounted to be the cause of the disease.

Many authors have described schizomycetes in cancer. Scheuerlin,[1] Koubasoff,[2] and Russell[3] have definitely asserted that they were the cause of the disease.

The labours of Shattock and Ballance (referred to later), of Senger[4] and Klein,[5] have shown these conclusions to be erroneous.

The work of Russell deserves more detailed notice. By staining sections of cancer with carbol-fuchsin, and after staining with iodine green, certain minute bodies ("fuchsin bodies") were picked out a bright red colour, and from their form Russell concluded that they were fission fungi. The drawing shown is taken from the author's paper, and represents a large cancer cell containing another, in the interior of which are four "fuchsin bodies." The intra-cellular cell is what later observers have described as an intra-cellular psorosperm, and the "fuchsin bodies" as spores. The accuracy of Russell's figures is now fully established; and though the "fuchsin bodies" have proved not to be fungi, Russell's earnest work has done much to stimulate inquiry into the nature of cancer.

In order to separate more or less the references to the work of authors who recognise sporozoa in cancers and other morbid tissues from those relating to the work of those who take the opposite view, the chronological order will be departed from.

Psorosperms have been described in this country in association with sarcoma.

W. B. Hadden[6] showed specimens from a case which he had diagnosed as multiple sarcomata. The sections were submitted to the late T. S. Cobbold, who stated that they were psorospermial nodules.

[1] Scheuerlin, 'Berlin Med. Soc.,' 1887.
[2] Koubasoff, 'Med. Cong. St. Petersburg,' 1889.
[3] Russell, 'Brit. Med. Journ.,' 1890.
[4] Senger, 'Berlin Med. Soc.,' Feb., 1888.
[5] Klein, 'Beiträge zur path. Anat.,' Bd. xi, p. 125.
[6] 'Path. Soc. Trans.,' vol. xxxiv, p. 237 (1885).

L. Pfeiffer[1] was the first to publish a description of parasitic protozoa in cancer.

On teasing out a fragment of melanotic cancer on the warm stage, Pfeiffer saw plasmodia in active amœboid movement, and cells resembling sporozoa. Pfeiffer compared the organism to the *Plasmodiophora brassicæ*, one of the microsporidia.

In an important work published in 1891 ('Die Protozoen als Krankheits Erreger,' Jena, 1891) L. Pfeiffer describes parasites as present in cancer, and expresses the opinion that in different cancers different sporozoa occur.

The peculiar bodies found in molluscum contagiosum had already by many authors, including Virchow (1865), been compared to gregarinidæ. Bollinger first strongly maintained that they were parasites, and compared them to a similar affection in birds. Neisser[2] and Pfeiffer[3] supported in detailed memoirs Bollinger's views, and attributed the disease to the parasites. Kaposi, Duhring, Török, and Tommasoli[4] regarded the bodies as products of degeneration, and could not discover in them the characters of coccidia.

A. Q. Silcock[5] published a case of acute psorospermosis associated with intestinal papillomata. A woman, aged 53, was admitted into St. Mary's Hospital under Dr. Broadbent in July, 1889, with fever and a swollen tender liver. Death soon followed. At the autopsy inflammatory areas and five papillomata with inflamed bases were found in the small intestine, and the liver was studded with pale spots which Silcock found to contain psorospermiæ somewhat resembling the rabbit's coccidia, save that they were much smaller. Cultivation gave a formation of lasting spores. The histology showed nodules surrounded by an inflammatory zone, and containing numerous amœboid psorosperms and swarm spore-cysts.

In 1889 R. Thomas[6] of Dorpat described within the nuclei of epithelial cells in cancers abundance of bodies resembling each other, and unlike anything met with in normal epithelial cells;

[1] 'Correspondenzblaetter des allg. aerztlich Vereins,' Thuringen, 1888.

[2] 'Vierteljahrschrift für Dermatol.,' 1888.

[3] 'Zeitschrift für Hygiene,' 1888.

[4] 'Monatshefte für Dermatol.,' 1890, No. 4.

[5] 'Path. Soc. Trans.,' 1889.

[6] 'Fortschritte der Medicin,' 1889, No. 11, June, p. 413.

unicellular organisms about 4—5 μ in diameter, highly refracting, and consisting of protoplasm, nucleus, and nucleolus. In other cases the parasite lay in a cavity immediately next the nucleus. The author recognised the resemblance of the cells to coccidia, but did not pronounce a definite opinion.

The origin of the subject in France is thus given by Wickham:[1]

Feb., 1889.—Darier was working out the histology of a case under Prof. Fournier. It had been diagnosed as "acne cornese." He discovered his "round bodies," and was certain that they were foreign elements. He suspected them to be parasitic without being able to decide the point. At the same time Wickham was investigating the histology of Paget's disease of the nipple, and had noticed the strange intra-cellular bodies, but without suspecting them to be parasites.

Meanwhile Darier, who was also "préparateur" at the Collège de France in the laboratory of Prof. Malassez, showed his preparations to Malassez, and pointed out his "corps ronds." Malassez expressed himself thus:—"Your preparations demonstrate to me not only that the 'corps ronds' are psorosperms, but also that these parasites are the cause of the disease, which is essentially an epithelial proliferation—an epithelioma if you will. Your specimens afford the finishing touch to a study I have been making since 1878 of epithelial growths, and I will show you my various sections containing similar bodies. I shall ask Albarran to show one of the cases at which I have worked. Until I saw your preparations I could not decide; now I am convinced."

May, 1889.—Albarran[2] showed his case of cancer of the jaw, and Malassez speaking on it said that he was familiar with the parasites since 1878, without being able to make certain of them until he had seen Darier's specimens.

The letter concludes, "The same day I saw Darier, and he said to me, 'Do you know what are the round bodies you have come across in Paget's disease? They are psorosperms, and probably the cause of the disease.' From that moment I worked at the psorosperms with Darier in Prof. Fournier's laboratory."

April 3rd, 1889.—Darier[3] read the results of his and Wickham's studies of the psorosperms of Paget's disease.

[1] In a private letter.

[2] 'Comptes rendus Soc. de Biol.,' 1889.

[3] Ibid.

August 8th, 1889.—Wickham published drawings and descriptions of the psorosperms of Paget's disease.

The affection which now rightly is termed "Darier's disease" has formerly been known as "acne cornee." It is quite distinct clinically from Paget's disease. Both patients, a man and a woman, were past middle age. The lesions were confined to the skin, and were very wide-spread. At first they consisted of papules formed by the accumulation of a substance resembling sebum in the mouth of the follicles; this was followed by some inflammation, and in one patient by epithelioma in the pubic region. The course of the disease was extremely slow.

Microscopically some of the cells of the rete were found to contain large clear cells with definite capsules. In the superficial layers these intra-cellular cells were replaced by dense, highly refracting grains.

Darier regards the disease as a psorospermosis, and the intracellular cells and bright grains as psorosperms. Darier also first described psorosperms in Paget's disease, and he was able to observe the process of sporing in cultures. Soon followed L. Wickham, whose observations have been embodied in a most valuable work, "A Contribution to the Study of Psorospermosis of the Skin, and of certain forms of Cancer."[1] Wickham's description of Paget's disease is complete in every way, and includes a full account of all the work that has been published on the subject since the individuality of the affection was first recognised by Paget in 1874. The author gives beautiful illustrations of the histology of his cases, and of another, Crocker's, in which a similar condition was present in the scrotum. He describes large encapsuled nucleated cells placed within epithelial cells, and encapsuled collections of spore-like bodies. The author regards these as psorosperms, and from their distribution in the tissues he considers them as the cause of the disease. Wickham looks on cancer as the third stage of the disease. The cancer may be of the squamous or of the tubular variety, and it contains psorosperms in great abundance. Wickham concludes that the cancer of Paget's disease is due to psorosperms, and that all cancers may prove to have the same causation.

Darier and Wickham's observations on Paget's disease have

[1] Paris, 1890.

been confirmed in England by Bowlby[1] and J. Hutchinson, jun.[2] Thin, in connection with Bowlby's paper, referred the cell-inclusions of Paget's disease to degeneration, an opinion with which Ruffer[3] and Walker have since expressed agreement.

Nils Sjöbring[4] was studying karyokinesis in sections of mammary cancers, hardened in alcohol and stained in Ehrlich's acid hæmatoxylin and carbolic eosin, when he observed many bodies identical in form with microsporidia, as figured by Balbiani and Bütschli. The bodies were stained more deeply by the eosin than the protoplasm of the cells which contained them. Some appeared to be within the nuclei, where they were, as a rule, surrounded by a space. Sometimes several were found within one nucleus. The larger parasites lay within the cell protoplasm, the nucleus being pushed aside. The parasites varied in size from 2 to 15 μ. They frequently contained chromatin filaments. Sjöbring regarded the bodies described by him as differing from those figured by Wickham.

Von Heukelöm[5] examined 200 cancers, and his observations agree in the main with Sjöbring's, as do also Kosinsky's.[6] The latter insists that the bodies in question exhibit evidences of a life-history which absolutely excludes cell degeneration as an explanation of their presence.

Without being able to express a definite opinion as to the nature of the bodies referred to, he agrees with Klebs[7] that the phenomenon regarded by many as spore-formation differs from accumulation and disintegration of leucocytes met with in ordinary inflammations.

Vincent[8] demonstrated in cancers bodies resembling coccidia, and described methods of staining.

Billroth[9] says that there is little doubt that molluscum contagiosum and the similar disease of birds are due to sporozoa. In

[1] 'Trans. Medico-Chirurg. Soc.,' 1890.

[2] 'Trans. Path. Soc.,' 1890.

[3] 'Journal of Pathol.,' vol. i, No. 2, p. 201.

[4] 'Fortschritte der Medicin,' 1890, July 15th.

[5] 'Centralblatt für allgemeine Pathol.,' 1890, No. 22.

[6] 'Ueber Physaliphoren in Krebsgeschwülsten,' Warsaw, 1890.

[7] 'Deutsch. med. Woch.,' 1890, No. 32.

[8] 'Comptes rendus Soc. de Biologie,' March, 1890.

[9] 'Ueber die Einwirkung lebenden Pflanzen- und Thierzellen,' Wien, 1890.

comparing tuberculosis and cancer he says it is clear that the sporozoa have the property of determining formative processes in animal cells.

The investigations of Shattock and Ballance[1] have been of the greatest importance; like Klein[2] and Senger, they show that Russell's position is untenable. They found careful cultivation experiments give entirely negative results. Incubation of fragments of cancer showed in sections wandering of nuclear matter. Also inoculation experiments with monkeys, dogs, and other animals, conducted with the utmost care and skill, gave always negative results. With admirable impartiality they review the work done on cancer up to the year 1891. And they relate Hanau's, Nerinsky's, Eiselberg's, and Hahn's successful inoculation experiments. They conclude that though cancer is in all probability an infective disease, the nature of the infection is still unknown.

In one of their communications ('Path. Trans.,' 1888) the authors delineate the histological appearances of a piece of human scirrhus after eight days' incubation. The bodies then drawn and termed nuclei have a great resemblance to amœboid psorosperms, and I think it probable that it shows a spore-formation on the part of parasites similar to that obtained by Darier in the "grains" he incubated from one of his patients. It is possible that the chromatin granules of these "nuclei" are the lasting spores of the sporozoa of cancer.

Walter K. Sibley[3] mentions an interesting case of human sarcoma successfully inoculated on a dog by C. O. Weber, and describes[4] sarcoma-like tumours (in a fish) caused by psorosperms.

Steinhaus[5] described the sporozoa-like bodies of cancer without expressing a definite opinion as to their nature. This author first advanced the theory of cell invagination to explain the presence of them.

A. Borrel[6] criticised chiefly the work of Darier and Wickham. He finds in sections of squamous epithelioma large encapsuled

[1] 'Brit. Med. Journ.,' 1891; 'Proc. Roy. Soc.;' 'Trans. Path. Soc.,' 1888, &c.

[2] 'Beiträge zur path. Anat.,' Bd. xi, p. 125.

[3] 'Trans. Path. Soc.,' 1891: "Non-contagiousness of Cancer."

[4] Ibid., 1890: "Psorospermia in relation to Tumour Formation."

[5] 'Virch. Arch.,' 1891, p. 533.

[6] A. Borrel, 'Archives de Médecine expt.,' 1890, vol. ii, p. 788: "Sur la signification des figures décrites commes coccidies dans les épithéliomes."

intra-cellular nucleated cells, which are sometimes replaced by a mass of nuclei apt to be mistaken for spores, and also "reticulated globes figured but not described by Wickham." These globes, according to this author, are due to a peculiar mode of degeneration. The intra-cellular cells are insufficient to justify the diagnosis of coccidia.

In a more recent work Borrel has modified his views, admitting that some cell forms in cancer are probably animal parasites.

Sheridan Delépine,[1] whose work on the psorosperms of the rabbit's liver has done so much to awaken an interest in and help in the formation of a sound basis for work on the pathogenic *rôle* of sporozoa, concludes,[2] "The evidences I have been able to collect are against the psorospermial nature of the bodies which I have observed in epithelial tumours, and I think that the same conclusions must also extend to the bodies observed in Paget's disease of the breast." In the last-quoted article Delépine describes within the epithelial cells of a human cancerous liver bodies resembling coccidia. Similar bodies have been described by myself in the ducts of cancerous breasts. In the drawing shown, which is copied by permission from Dr. Delépine's article, "one of the cells and perhaps two others contain besides the nucleus a body which seems encapsulated, and which in the fresh state closely resembled young psorosperms."

E. Klebs[3] opposes the view that cancerous tissue contains sporozoa, and considers the bodies described as such to be blood-corpuscles, products of degeneration, &c.

Firket[4] and Schütz[5] have taken up a position similar to that of Klebs.

Cornil[6] considers that karyokinetic processes have been mistaken for spore-formation.

Hansemann[7] has found irregular mitoses in epithelia other than those of cancer, and from the fact that the irregular processes of cell-division and "so-called" spore-formation are chiefly met with

[1] 'Trans. Path. Soc.,' vol. xli, 1890; vol. xlii, 1891.
[2] 'Trans. Seventh Internat. Congress of Hygiene,' London, 1891, p. 93.
[3] Klebs, 'Deutsch. med. Woch.,' 1890, No. 32.
[4] Firket, 'Centralblatt für allg. Pathol.,' 1890, No. 20.
[5] Schütz, 'Münch. med. Woch.,' 1890, No. 35.
[6] Cornil, 'Journal de l'Anat. et de Phys.,' 1891, No. 1.
[7] Hansemann, 'Virch. Archiv,' 1891.

in the degenerated parts of cancers, suggests that they are due to want of nutriment. This observer agrees in the main with Cornil.

Fabre-Domergue[1] identifies the bodies described as sporozoa, some with those described by Virchow as cells of endogenous origin, others with bodies described by the same author as degeneration products.

Duplay and Cazin,[2] summing up their investigations on the work of Nils Sjöbring and others, regard the parasitic nature of the bodies as not proven.

G. Hauser[3] discusses the bacillus theory. The author regards the identity of the structure of the secondary growths wherever occurring to contradict the bacillary hypothesis.

Kiener,[4] Professor of Pathology at Montpellier, has expressed himself as sceptical, and influenced Borrel's first article.

Ribbert[5] could not detect the crooked bodies described as spores by Nils Sjöbring. Ribbert comes to the conclusion that the bodies are due to a metamorphosis of the nucleus, the granular bodies being the remains of nuclei or portions of chromatin.

Virchow[6] had not been able to observe anything in molluscum contagiosum or in cancer which pointed to a parasitic origin.

Another supporter of the view of degeneration may be taken, Welch, of the Johns Hopkins Hospital, quoted by Nœggerath as coming to the following conclusions:—That the parasitic nature of the cell-inclusions is not proved, but, on the contrary, they are of the following characters: (1) portions of cells keratinised centrally; (2) masses of kerato-hyalin; (3) included leucocytes with degeneration or fragmentation of nucleus; (4) fragments of nuclei of leucocytes.

[1] Fabre-Domergue, "Les Coccidies dans les Néoplasmes," 'Congrès de Chirurgie,' Paris, 1891.

[2] Duplay and Cazin, 'Trans. Seventh Internat. Congress of Hygiene,' vol. ii, p. 81, London, 1891.

[3] G. Hauser, 'Das Cylinder Carcinom des Magen und Dichdarms,' p. 124, 1890.

[4] Kiener, referred to by Borrel, 'Arch. de Médecine expérimentale,' vol. ii, 1890, p. 893.

[5] Ribbert, 'Deutsch. med. Woch.,' 1891.

[6] Virchow, in a note on Steinhaus's article, 'Virchow's Archiv,' No. 127, p. 188, 1892.

Strœbe[1] admits the close resemblance of some of the cells of cancer to sporozoa, but leaves open the question of identity.

Ramsay Wright,[2] like Russell, thinks the bodies described as sporozoa belong to the saccharomycetes.

E. Nœggerath[3] has ably reviewed the whole subject in a work to which I am greatly indebted. He confirms all the descriptions given by Nils Sjöbring, and up to a certain point was prepared to entertain the conclusions of the latter; but in a section of cancer, stained first with carmine and then by Gram's stain, he found an appearance as if part of the nucleus was undergoing liquefaction, and concluded that most of the intra-cellular bodies arose in this way, and were not parasites. Some of the cell-inclusions, however, resembled protozoa closely, and the author is prepared to admit them as such. But these forms are so few that they cannot have any important *rôle* in cancer. He observed the subdivision of large cells in cancer, and the migration of the resulting particles —the swarm-spore formation. Reference is made to Ehrlich's observations on the erythrophile and cyanophile portions of the nuclei of cells. The work is one of the most valuable published on the subject. The illustrations are excellent. Many will feel that, had the author been more practised in biology, he would have come to a different conclusion.

Podwyssoski and Sawtschenko[4] give complete references to previous work, and describe, with drawings, parasites which they found most abundantly in mammary cancers. The forms are the encapsuled intra-cellular cell, the conglomerated masses of minute cells (spores), and small non-nucleated cells within epithelial cells, adherent to the nucleus. They thought that these forms warranted the positive conclusion that they were parasitic sporozoa, but they could not from the histological characters alone decide as to whether they were the ætiological factors of the disease.

Later, Sawtschenko[5] traces the life-cycle of the parasites in a case of cancer of the lip. He finds evidence that the capsules are

[1] Strœbe, 'H. Ziegler's Beiträge,' 1891.

[2] Ramsay Wright, 'Centralblatt für allgemeine Pathol.,' 1890, No. 11.

[3] Nœggerath, 'Beiträge zur Struktur und Entwickelung des Carcinoms,' Wiesbaden, 1892.

[4] 'Centralblatt für Bacteriologie,' &c., 1892, xi, Nos. 16—18.

[5] Ibid., July 5th, 1892; 'Weitere Untersuchungen über schmarotzende Sporozoa in den Krebsgeschwülsten,' 1891.

secreted by the parasite and not by the host-cell, and is inclined to regard the parasites as the cause of the disease. His illustrations refer chiefly to the sporing process.

Soudakewitch [1] gives detailed descriptions of cell-inclusions he met with in ninety-five cancerous growths, with three plates. These cell-inclusions, the author found, could not possibly be the result of degeneration. He found them most abundantly in cancer of the liver. The appearances described led the author to say that in every case parasites of the nature of sporozoa were present.

Metchnikoff,[2] in a note on Soudakewitch's manuscript, expresses the decided opinion that the cells met with and described by Soudakewitch are parasites akin to the rabbit's coccidia.

In a later article Soudakewitch [3] continues his work and gives additional figures, which, like those of the first article, are of great beauty, and after having verified almost every one of his descriptions I can also say of complete accuracy. Soudakewitch regards the parasites as sporozoa, and thus describes the liberation of swarm-spores :—"The destruction of the cancerous cells is followed by the liberation of the parasite of which the capsule ruptures, and the spores, escaping, infest the neighbouring cells."

G. Sims Woodhead, in the "Morton Lecture on Cancer" of 1892 ('Lancet,' May 7th, 1892), describes encapsuled cells, some with a stellate arrangement of protoplasm situated within epithelial cells of cancers, and closely resembling coccidia. The author also alluded to Soudakewitch's observations, and mentioned that in some sections submitted to him by Ruffer and Walker he found bodies with all the characteristics of coccidia, and absolutely distinguishable from blood-corpuscles and products of degeneration. "In some of the capsules there appears to be a process of division going on, whilst here and there are capsules in which several (four to twelve) small rounded or crescent-shaped psorospermiæ,[4] in other respects like the coccidia except that they have no capsule, may be made out. In one or two instances these appear to be lying free between the epithelial cells, from which

[1] 'Annales de l'Institut Pasteur,' tome vi, No. 3, p. 145.

[2] Ibid., p. 158.

[3] Ibid., No. 8, Aug. 25th, 1892.

[4] Sims Woodhead has since ('Brit. Med. Journ.,' 1893) stated that this observation was erroneous, without giving detailed explanation.

they have probably escaped on the breaking down of the mother capsule. They are of various sizes, from the diameter of a red blood-corpuscle to three or four times that size. . . . We are still very far from the proof that these organisms are the actual cause of cancer."

In September, 1892, at the International Dermatological Congress held at Vienna, a discussion of the nature of Darier's disease and molluscum contagiosum took place.

Boeck (Christiania) thought the peculiar bodies met with in Darier's disease were the result of hyperkeratosis.

Neisser (Breslau) thought Wickham's conclusions with regard to Paget's disease untenable; he thought Darier's disease had much in common with molluscum contagiosum, and the latter he had no doubt was due to coccidia. The author showed preparations in which spores were visible.

Ehrmann (Vienna) had found in pemphigus foliaceous cells similar to those described as coccidia in cancers. He thought it utterly impossible that two so different diseases could be evoked by the same cause, and therefore he regarded the psorosperm-like bodies in these diseases as degeneration products from epidermal cells. He shared Neisser's views with regard to molluscum contagiosum.

Ianovsky (Prague) related a case of Darier's disease in which healing took place in some parts whilst extension was going on in others.

Schwimmer recommended energetic cauterisation in the treatment of the disease.

Török explained the position taken up by Tommasoli and himself. On account of the failure of cultures, and chemical and anatomical differences between them and rabbits' psorosperms, they supported the cell-degeneration theory. The bodies resembling psorospermia in Paget's disease were nothing more or less than epithelial cells enormously increased as the result of inflammatory proliferation. A number of mitoses were "giant mitoses." Török regarded the psorosperm-like bodies in cancer as epithelial cells and white blood-corpuscles.

Jackson Clarke[1] published a case of psorospermosis of the urinary tract, giving references to similar cases by Virchow, Lindemann, Eve, Bland Sutton, and Targett. The article is illus-

[1] Jackson Clarke, 'Path. Trans.,' 1892, p. 94.

trated, and reasons are given for regarding certain bodies as parasites belonging to the "sporozoa."

Foa at first declined to accept many of Soudakewitch's figures, but in a later article[1] describes sporozoa which form swarm-spores, which on escaping from their place of origin enter at once into fresh epithelial cells.

M. Armand Ruffer and J. Herbert Walker, in October, 1892, in 'The Journal of Pathology,' vol. i, No. 2, publish an investigation on some parasitic protozoa found in cancerous tumours. The paper is accompanied by references and illustrated by beautiful plates. The encapsuled intra-cellular stage of the parasite is almost exclusively dealt with in this valuable paper. These authors considered the bodies described by others as spores or amœboid psorosperms to be the products of degeneration. In a previous paper[2] the same authors published some drawings which are reproduced in their second article. These drawings in their first communication represent bodies which the authors term parasites. The same drawings are done in colours in the second article, but the bodies represented are said not to be parasitic. No explanation of this change of opinion is given. The authors state no reasons for regarding the bodies in question as protozoa beyond mentioning the remarks of Sims Woodhead already referred to, and quoting Metchnikoff's statement, "They have, indeed, all the characteristics of coccidia." But neither the illustrations nor the text give any proof that the bodies do possess all the characters of coccidia, for no kind of spores are described. The bodies regarded by previous authors, *e. g.* Podwyssoski and Sawtschenko, as swarm-spores or amœboid psorosperms are described as products of degeneration (see pl. xvi, fig. 31).

In reviewing Virchow's observations they do not state which of his figures they take to represent parasites, and which to represent "cells of endogenous formation."

In noticing the work of Podwyssoski and Sawtschenko they say, "We have little doubt that the bodies described by these authors were probably simply degenerated cancer cells." They do not refer individually to the drawings published by the Russian observers, two of which certainly represent bodies of the same nature as that represented by Ruffer, fig. 6, pl. xiv, as a parasite.

[1] Foa, 'Centralblatt für Bacteriol. u. Parasitenkunde,' Aug. 9th, 1892.

[2] 'Brit. Med. Journal,' July, 1892, p. 113.

The admirable work of Wickham is dismissed by quoting the opinion of Borrel to the effect that the bodies drawn by Wickham were products of degeneration. This is all the criticism the authors give of the French observer's work, although in the next sentence they state that Borrel had later reconsidered and modified his opinion.

The authors use the wide term *Protozoa* to designate the parasitic organisms, though all previous observers had recognised them definitely as "*sporozoa*." This failure to grasp the nature of the process of swarm-spore formation in the sporozoa of cancer may in part be due to their having employed the mixture of aniline dyes known as the Ehrlich-Biondi stain, which, though most useful to demonstrate the parasites when once their nature is understood, is not so reliable as acid hæmatoxylin and eosin for giving an understanding of their nature.

The authors make frequent mention of invaginated cells, and they illustrate the condition (figs. 35—37, pl. xvi). I consider that these illustrations are unsatisfactory because the so-called invaginated cells are stained quite differently from those within which they lie; whereas had they been of similar nature, *i. e.* epithelial, it is to be presumed they would have stained in somewhat similar manner. Both this question of invaginated cells and that of phagocytosis in cancer require, in my opinion, much more study. One of Ruffer's illustrations (pl. xv, fig. 17) is of considerable interest. It represents the atrophy of epithelial cells owing to the pressure of cicatricial tissue. I believe this process takes place in atrophic cancers and in atrophying parts of growing cancers, and also phagocytosis under the same conditions; but in actively growing parts of cancers and sarcomas I have convinced myself that the opposite conditions prevail, namely, destruction of fibrous tissue and of leucocytes by amœboid psorosperms.

Ruffer and Walker make no mention of parasites in sarcoma.

This paper is deficient in the important matter of fair criticism of work previously published by other observers, and is retrograde in the sense that it is behind the work of many previous authors who recognise the parasites as sporozoa.

Argument.—It will be at once perceived that there is considerable difference of opinion as to the interpretation that should be given of certain cells met with in cancer, rodent ulcer, Paget's disease, Darier's disease (acne cornee), and molluscum contagiosum.

Biologists such as Balbiani, Metchnikoff, and others all regard them as sporozoa. The group of sporozoa include many parasites, gregarinidæ, coccidia (Leuckart), and a great number of the hæmatozoa of man and animals, as has been shown by Laveran, Danielewsky, Golgi, and others. Many pathologists also hold the opinion that the atypical cells of cancer, molluscum, Paget's and Darier's diseases are parasitic sporozoa.

It will not be out of place here to recall the similar sporozoa met with in the rabbit's liver and intestines, described as pus cells in 1820 by Hake, and by all now accepted as parasites. Leuckart[1] created for them the class Coccidia, the name being derived from the encapsuled spores which under certain circumstances form within the definite capsule of the large parasites. Leuckart came to the conclusion that the parasites do not multiply within their host, but only after being separated along with the fæces. Sheridan Delépine[2] in a comprehensive description showed conclusively that the parasites multiply in the body, and that in the adenomatous tumours of the rabbit's liver the encysted spores are not formed. I have been able to verify Delépine's observations. The amœboid cells cluster upon and enter the epithelial cells, where they grow, and when they reach large dimensions they escape and lie free in the cyst, where they form numerous amœboid (unencapsuled) spores, which return to the epithelium and begin the round again. Sometimes the psorosperms multiply rapidly in rabbits, and a fatal termination is often the result; as a rule, however, tumours of the character of cystic adenomata are formed.

The gregarinidæ of earthworms, the coccidia of silkworms, snails, fishes, mice, and other animals, resemble closely the coccidia of the rabbit.

Laveran was the first to find a plasmodial parasite in the blood of persons suffering from ague. That plasmodium is identical in general structure with the amœboid psorosperm. Parasitic hæmatozoa have been found in the blood of many kinds of animals. In some of them the sporing process has been traced, and it is almost identical with that found in the peculiar cells of cancer and other morbid formations. Some of these parasites of the blood at a certain stage are studded with granules of pigment. In the same way I have found the parasites of some cancers and sarcomata

[1] 'Parasites of Man.'

[2] 'Trans. Path. Soc.,' 1890.

become pigmented; thus I would explain the peculiar character of those growths.

Are, then, the anomalous cells met with in various human growths parasites akin to these sporozoa? Before this question can be answered the evidence must be fairly digested.

Time allows only the briefest examination of evidence if, as I hope, a discussion is to follow.

It may be seen on reviewing the opinions given above that they may be classified thus:

1. Those of authors who refer the appearances of cells resembling parasites in cancer to abnormal cell multiplication, or degeneration.

2. Those of authors who, recognising the probability that parasites will be found in cancer and sarcoma, regard their presence as not yet proved.

3. Those of authors who find sporozoa present in cancer, but do not find complete evidence of a causal relationship between the parasites and the growth.

1. According to the first group of observers, a small reticular mass of protoplasm, at first included in or adherent to the nucleus, enlarging to a definite cell, and often becoming encapsuled, and exhibiting an exuberance of biological activity in the shape of giant mitosis, irregular mitosis, and spore-like subdivision, may be conceived to be the erratic offspring of tissue-cells under the stimulus of some abnormality of nutrition. This view, however, even if restricted to remnants of embryonic tissue according to Cohnheim's hypothesis, would imply such an instability of animal structure as would be incompatible with the persistence of species. Indeed, I think a close study reveals the fact that the spore-like amœboid cells are at first formed outside the tissue-cells, and afterwards enter the cells in the manner of parasites. Their morphology is that of sporozoa, and a comparison of all the parasitic sporozoa known in the various orders of animals with those of man is, I think, conclusive as to their nature.

Borrel's account of squamous epithelioma may fairly be taken as representative of the hypothesis of degeneration. Many authors besides myself have shown that the appearances are not degenerative, but are due to active biological processes. The "reticular degeneration" of Borrel might be due to a process akin to coagulation. Careful drawings and descriptions of the stages subsequent

to the formation of a reticular mass show, however, that this is not the case.

2. Amongst the many authors who have expressed opinions that the presence of sporozoa or even of any protozoa is doubtful, the work of Shattock and Ballance commands the most respectful attention. Their many negative transplantation experiments are, however, more than counterbalanced by the positive results obtained by Hanau and many others. It is to be gathered from the labours of Shattock and Ballance that the sporozoa of man cannot flourish in animals to any appreciable extent, but their failure to transplant coccidia from one part of the same rabbit to another part of its body shows that no importance is to be attached to the results of transplantations as far as the argument for and against the parasitic nature of cancer and sarcoma is concerned. To any observer who has not fully appreciated the morphological identity of the sporozoa with the varied atypical cell forms met with in the human growths under consideration a sceptical attitude is the only one possible.

In no single instance have bacteriological methods been successfully applied to the study of any of the recognised parasitic sporozoa. And when it is remembered that these animals pass one stage of their existence, not only within, but in the closest physiological dependence on, cells of other animals, the difficulty of making successful cultures must be realised.

I may here be allowed to quote from a recent article from the pen of Laveran.[1]

"The fact that neither cultivations of the parasite of paludism in artificial media, nor inoculations of animals with the blood of persons affected with the disease have been successful, has been used as an argument against the existence of the parasite. The argument might avail if schizomycetes were in question. . . . One cannot cultivate the *Filaria sanguinis-hominis*, and yet no one has ventured to contest the parasitic nature of that affection."

3. I may now consider the last class of opinions, namely, those which pronounce parasites to be present and yet do not see that the causal relationship between parasite and disease is established. The published work of Ruffer and Walker[2] may be taken as an example. The beautiful plates by Metchnikoff and Ruffer with this

[1] 'Arch. de Médecine expérimentale,' vol. ii, 1890, p. 1.

[2] Loc. cit.

article illustrate well one stage of the parasite's existence. But the all-important process of sporing is only mentioned to be rejected, and the amœboid stage not recognised, though it is figured by Ruffer (pl. xvi, fig. 31) as a product of degeneration; and, if I mistake not, the keen and learned eye of Metchnikoff has for once erred in representing (pl. xiv, fig. 15) spore-formation as an example of phagocytosis. Indeed, I fail to see how the parasite can be relegated to the group of sporozoa if the sporing stage is not recognised.

Of all previous writers Sawtschenko[1] most closely agrees with my own observations, and this author is inclined to attribute the causation of the disease to the parasites. As far as I am aware I am alone in finding in cancers and sarcomas of varied kinds unmistakable evidence of what I consider to be the essential factor of the malignant tumours, namely, the streaming of amœboid psorosperms into the tissues beyond the growth. The amœbæ and large plasmodia digest these tissues and awaken inflammation, so removing resistance and enabling the growth to infiltrate the softened parts. The same process causes detachment of fragments of cancer or epithelioma to be carried along the lymph and blood-currents, and so to form secondary growths.

I have had recently an opportunity of studying one of Darier's cases in life, and of reading an as yet unpublished article by this author. I am convinced that he is right in regarding acne corneæ, or, as it is now rightly called, "Darier's disease," as a psorospermosis.

I am fully convinced by Wickham's admirable work that Paget's disease and the cancer which usually supervenes on it are caused by psorosperms. I am convinced by the work of Bollinger, Neisser, and others, and by my own observations, that molluscum contagiosum is a psorospermosis, in spite of Török and Tommasoli's chemical tests.

I have fully convinced myself that a cystic condition of the urinary tract—that at least some adenomata of the kidney, some adenomata and some non-cancerous cysts of the breast, and carcinomata and sarcomata—are due to parasitic sporozoa, which for the present may be termed psorosperms.

I may ask even the most sceptical to accept this as an hypothesis. Then what a field of possibilities presents itself to the mind!

[1] Loc. cit.

What a cohesion in the pathological department of biology! The severer diseases of earthworms and silkworms are almost identical with those of man.

To look at the practical bearings of the matter.

The general indications are for more investigation. The life history of the parasites must be completely worked out. Some may live in marshy places, like the paludal hæmatozoa, others may exist normally in the moist parts of our bodies and become noxious only under certain circumstances.

Renewed investigations may reveal their presence in many other diseases besides those with which they have already been found to be associated. The adenomata, cancers, and cysts of the thyroid may prove to be due to parasites which infest the waters of goitrous districts. Syphilis may be found to be due to an animal parasite.

In the course of practice more careful examination of cyst-contents and of the histological structure of growths will be necessary for complete diagnosis and correct treatment. The difference between some cystic diseases, adenomata and cancers, is, it appears to me, due to differences of resisting power in the individual attacked, not in the cause of the disease. More prompt removal of simple tumours is clearly indicated. The result of the work of Raymond Johnson and Cecil Beadles on the non-cancerous parts of cancerous breasts now receives its full explanation, and their conclusions complete confirmation. Since we have in quinine and arsenic drugs which have been proved adequate to check the activity of the sporozoa of paludism, there may yet be found drugs capable of arresting the activity of the sporozoa of cancer and sarcoma. It has been shown especially by Councilman and Lafleur that some forms of dysentery are due to amœbæ. I have fresh in my memory the case of a young man who shortly after an attack of dysentery died of rapidly fatal cancer of the liver. It may be the same parasite caused both affections. Instances might be endlessly multiplied. I will only ask permission to emphasise the fact that in the study of the pathogenic sporozoa there is a splendid field for the highest form of medical industry, namely, preventive medicine.

February 21*st*, 1893.

2. *Observations on parasitic organisms in carcinoma.*

By JAMES GALLOWAY, M.D.

DR. GALLOWAY said that he did not wish to detain the Society long, as he had recently fully explained his views on this subject.[1]

He proceeded to demonstrate by means of microphotographs the only appearances which had yet been described in carcinoma which might be considered to be of parasitic origin. These bodies occurred in almost all cases within the cell wall, situated either in the cell protoplasm or in the nucleus. They were usually spherical, consisting of a capsule and a nuclear substance; from the capsule inwards, and also outwards from the nucleus might be seen occasionally radial striations. They occurred usually singly in the cells, but groups of as many as eight or ten of smaller size might be seen in certain specimens. Their size varied from about 2 μ up to 12 μ in diameter.

The appearances he considered to be the same as those described by Soudakewitch, Ruffer and Walker, and by certain other observers.

The speaker then proceeded to mention certain appearances in tissues which were at times misleading; these he mentioned under the headings of—

1. The partial invagination of one cell into the cell body of another.

2. The inclusion of leucocytes within epithelial cells.

3. The endogenous division of cells, with the necessary inclusion given rise to thereby.

4. The inclusions brought about by irregular mitosis, a phenomenon very common in carcinomatous epithelium.

5. The various appearances produced by the already well-recognised degenerations of cells in carcinoma—colloid, keratinous, &c.

The most serious criticism which had been brought against the hypothesis that the structures which had been described were parasitic, was that the appearances mentioned were due to irregular mitosis occurring in nuclei, by means of which portions of chromatin, broken off the main chromatin mass, were enabled temporarily to gather a border of protoplasm around them while

[1] 'Morton Lecture,' 1893, Royal College of Surgeons, England.

still within the mother-cell wall. This view had recently been taken by several observers in German laboratories.

In the speaker's opinion this hypothesis was not adequate to account for the appearances which he had described at the commencement of his observations. On the other hand, their occurrence within the cell as a distinctly foreign substance, their appearance, so sharply suggestive of an organised structure, the staining reactions which they give, so distinct from those presented by the normal contents of cells, their analogy in this and other respects to already recognised species of protozoa existing in lower animals,—all point forcibly to the conclusion that these bodies are parasitic organisms in cancerous epithelium.

In reference to the remarks made by Mr. Jackson Clarke, the speaker said that he already had criticised his observations adversely. He had hoped that Mr. Clarke might have reconsidered both his observations and conclusions, with results differing from those he had reiterated this evening. Of the specimens shown to the Society by Mr. Clarke he had seen none that might not be accounted for by the existence of cells either normal or degenerate in character. In those specimens, and in the photographs thrown on the screen by Mr. Clarke, if all that he declared to be "psorosperms" were really such, there would be little tissue left, either normal or diseased. These appearances the speaker considered to be leucocytes, or the epithelial cells of carcinoma in a more or less degenerate condition. *February* 21*st*, 1893.

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3. *On protozoa and cancer.*[1]

By M. Armand Ruffer.

[With Plate V.]

The origin of every scientific discovery is generally wrapped in a good deal of obscurity, and this is markedly the case in this question of the parasitic bodies found in cancer. The observers

[1] At a meeting of the Society held on March 7th I criticised the statements and preparations of another member of the Society. As I understand that his description of those preparations will not be printed in the 'Transactions' my criticisms are also omitted, although I have no reason to alter the opinions then expressed. At the same meeting I gave a short account of Mr. Plimmer's and my researches on the multiplication and staining reactions of the parasitic organisms of cancer. Since that date we have obtained confirmatory evidence of our observations, have extended them in other particulars, and have demonstrated

who first described in cancer structures which they assumed to be parasites were Thoma in Russia, and Malassez in France; but I am unable to say whether these observers have really seen the same bodies as ourselves or not, as they neglected to illustrate their papers either by drawing, painting, or photography. Shortly afterwards we have the papers by Darier, and Wickham, both of whom described coccidia in cancer. I have, in another place, criticised the work of these observers, and as I shall come back to this point, it will be sufficient for me to say that the bodies described by Darier and Wickham were but cancer cells more or less altered, leucocytes, &c., and certainly not parasites. I need not refer except in passing to the work of Nils Sjöbring, E. Siegenbeck van Heukelöm, Hauser, Russell, Shattock and Ballance, Klien, Cazin, Stroebe, Schütz, L. Pfeiffer, Ribbert, Cornil, Fabre-Domergue, Bowlby, J. Hutchinson, Thin, Delépine, Borrel, Steinhaus, Vincent, Welch, Sims Woodhead, and Noeggerath, who wrote on this question ;[1] but must draw attention to a paper by Soudakewitch, who, it appears to me, was the first to clearly recognise the parasites of cancer. Since that time, Ruffer and Walker, Sawtschenko, Foà, Borrel, Plimmer, Galloway, and others have recognised similar bodies as parasites, though their existence as such has been strenuously denied by Delépine, Boyce, and others.

In all scientific questions the methods used are of the greatest importance, and nowhere more so than in histological investigations on pathological structures. In the first place, the method of fixation must be carefully chosen. Our first observations were made with cancers fixed in alcohol, and there is no doubt that occasionally beautiful results can be so obtained; but this method has the great disadvantage of causing extreme shrinking of the tissues and rendering the smaller parasites almost invisible, and this fact soon induced us to give up its use. Sublimate solution, of which we spoke at first with too scanty praise, nevertheless proved valuable afterwards, and our first unfavorable results were no doubt due to a technical mistake on our part,—insufficient washing, to wit. The aqueous solution must be saturated, and the pieces, which ought not to exceed a few millimetres in thickness,

these modes of division to our fellow-workers at the laboratories, to Drs. Metschnikoff and Roux of Paris, and many other competent pathologists.

[1] Those interested in the subject will find in my first paper, 'Journ. of Path.,' No. 2, 1892, the complete bibliography on this question. An interesting paper on the same subject has quite lately been published by Dr. Cattle of Nottingham.

remain in it for twenty-four hours; they are then washed for the same period *in running water*, next hardened in 30 per cent., 60 per cent., and 90 per cent. alcohol, remaining in each for twenty-four hours; and are lastly placed in absolute alcohol for one or more days until required for cutting.[1] Normal saline solution saturated with sublimate, Müller's fluid saturated with the same salt, Flemming's solution, Heremann's solution, 1 per cent. osmic acid solution, and chromicised spirit, may each be used successfully for fixing purposes, but the pieces hardened in all of these (the last excepted) must always be well washed in running water for twenty-four hours at least before being placed in weak alcohol. These methods of fixation, which are now in common use in all histological laboratories, have not sufficiently attracted the attention of pathologists; and we have been astonished at seeing sections of cancer prepared by teachers of pathology, in which, owing to the deficient methods used for fixing, hardly a single anatomical structure had its normal appearance. How, under such conditions, was one to recognise such delicate structures as protozoa, when the nuclei of the cells were but poorly defined and the protoplasm had shrunk almost to nothing?

Similarly with cutting and staining the sections. For cutting purposes, nothing is better than soaking in paraffin, but for staining we have a variety of methods which will give good results. To get typical preparations one must not lose sight of the fact that if parasites are to be distinguished from nuclei, &c., we must double or treble stain the section with a nuclear and one or more protoplasmic dyes; for, as I have repeatedly pointed out, the specific parasites of cancer have a special affinity for the latter class of dyes.

A great deal in the recognition of these parasites depends on their staining reactions, just as in bacteriology, in pathology, and even in chemistry, some of the best tests at our disposal are colour tests; and this being the case, it is necessary, when attempting to illustrate any paper on advanced histology, to supplement the text by paintings, and not simply by drawings or photographs. The man who trusts to photography in such matters is like the man who would go to a picture gallery in smoked spectacles, or who would judge of a picture by a photograph of it. Photography, especially, is often

[1] A useful modification is to abstain from washing in water and place the sections when cut in tincture of iodine for thirty minutes. Wash in spirit and afterwards in water. Care must be taken to use very small pieces of tissue, and to leave them three or four hours only in sublimate.

the very worst kind of pictorial representation of sections, for when one looks down the microscope, one does not look at one plane only of the object, but at several planes in succession, the final opinion being formed from the synthesis of the pictures seen. A photograph is only too often the picture of an object as it would be seen by a person who had lost the power of accommodation. Hence I have always preferred illustrating my papers with coloured drawings, although, thanks to the kindness of many of my friends, especially of my friend Plimmer, I am in possession of many excellent photographs of my sections. If we look, for instance, at the photograph in Plate V, it would be possible to argue that the parasite undergoing division in Fig. 2 was simply a particle of chromatin separated from the nucleus; but if we examine the preparation, this latter supposition is at once proved to be erroneous, for the simple reason that the nucleus of the cancer cell has taken the methyl-green intensely, whilst the two nuclei of the parasite are red from the fuchsine contained in the Ehrlich-Biondi reagent. Moreover we find that several details plainly visible with the microscope are invisible in the photographs, because they are at a slightly different level. Far be it from me to minimise the use of photography in microscopic work, but I would simply point out that it is unscientific to say that this question must be settled by photography, when at present the photographs of sections, even when made by the most skilled sun-picture specialists, are, with high powers of the microscope, but caricatures of the reality. And when in addition to this fact we remember that those who rely on photography for their supposed proof of the supposed cellular derivation of the structures which we regard as parasites, have generally taken their pictures from cancers fixed with unreliable and obsolete reagents, it will be evident that we have not been working under the same conditions, and therefore that the results must of necessity be different.

Having in another paper, written in conjunction with my friend Plimmer, given a full account of our researches on the structure and multiplication of these parasites, I need not here enter into that question at any great length, more especially as, thanks to the courtesy of this Society, I am able to place before the readers of their 'Transactions' a few pictorial representations of the structures which we regard as parasitic, together with some of the forms of multiplication which we have observed. A few comments may perhaps not be out of place in order to prevent misconceptions,

DESCRIPTION OF PLATE V,

Illustrating Dr. M. Armand Ruffer's communication on Protozoa and Cancer. (Page 209.)

(All the figures are taken from carcinomata of the breast.)

FIG. 1.—Fixed in Flemming's solution. Methyl-green and Biondi-staining. Cancer cell with parasites in its interior. The radiation is well marked; small chromatine granules are scattered through the parasite's protoplasm. Beck, oc. 1, obj. immers. $\frac{1}{12}$.

FIG. 2.—From the same cancer. The parasite has just divided into two. The division is not completed. Fixing, staining, and magnification as in fig. 1.

FIGS. 3, 4, 5, 7.—Fixing in chromicised spirit. Hæmatoxylin and cochineal stain. Verick, oc. 1, obj. immers. $\frac{1}{12}$.

FIG. 6.—Fixing, staining, and magnification, as in figs. 1 and 2. Notice that the parasite has two nuclei, but the signification of the smaller one is unknown.

FIG. 8.—Fixing in chromicised spirit. Eosine and aniline-blue staining. Two typical parasites stained blue. Verick, oc. 1, obj. immers. $\frac{1}{12}$. Figure projected on the table.

FIGS. 9, 10.—Fixing in chromicised spirit. Gentian-violet and acid-fuchsine staining. Parasites have taken the red stain. Magnification as before.

FIG. 11.—Fixation in chromicised spirit. Aniline-blue staining. Division and regular segmentation of the parasite. Magnification as before.

FIG. 12.—Fixation in osmic acid. Hæmatoxylin stain. A bunch of parasites showing well-marked metachromatism. This probably represents the separation of the parasites after segmentation. Magnification as before.

FIGS. 13, 14.—Fixation in chromicised spirit. Hæmatoxylin and cochineal staining. Segmentation of parasites. Magnification as before.

FIG. 15.—Section of carcinoma of breast. Gentian-violet and acid-fuchsine staining. Nuclei of parasites red. Verick, oc. 1, obj. immers. $\frac{1}{12}$.

Fig. 1

Fig 6

Fig 11.

Fig 8

Alice M. Ruffer, del,

especially as, since the beginning of our studies on this question, my collaborators and I have discovered new facts which to some extent have caused us to modify our opinions.

In the first place, in our former paper we stated that the parasite did not occur in the nucleus of the cell, and this is the case for the large majority of sections; but in a few of them the parasites undoubtedly occur in the nucleus, and may even divide and multiply inside it. And I may take this opportunity of stating that the word "spore," which I, and not my collaborators, used in order to denominate such young parasites, was inappropriate, as up to the present there is no evidence to show that these bodies are true spores. In osmic acid preparations, and even in other preparations, it is possible to stain such structures with hæmatoxylin; nevertheless they always have special affinities for certain protoplasmic aniline dyes—for orange and acid fuchsine, for instance. They also frequently display the phenomena of metachromatism. It would be absurd to suppose that the parasite, even when full-grown, is absolutely unstainable with hæmatoxylin, for any staining reagent will stain any animal tissue if only allowed to act long enough; but then by that time the entire preparation would be saturated with the stain, and all possibilities of differentiation would have vanished.

I have called "nucleus of the parasite" the central body which stains more darkly than the rest; but it is exceedingly probable that this central point is in many cases really but part of the parasite's nucleus, the nucleolus possibly, just as in the coccidia which infest rabbits, the only part of the nucleus which takes up the colouring matter easily in hardened specimens is the nucleolus. The study of the nucleus of protozoa in sections of hardened specimens is notoriously difficult, and nothing can be definitely said about it until it has been more extensively observed in the living state.

With regard to the multiplication of the parasite, Walker and I were unable to see it in our first series of sections, but since that time Plimmer and I have followed its various stages. I was under the impression that these parasites multiplied by the formation of spores inside a cyst, and spent much time in searching for these spores; but although I would not deny the possibility of such spore-formation in the parasites of cancer, yet I must confess that I have never seen it. On the other hand, I think Plimmer and I have clearly established that such parasites multiply by fission into

two or multiples of two, the division and subdivision of the parasite being accompanied or followed by the division of the capsule. In this way a kind of cyst, or rather an agglomeration of parasites, is formed, which may consist of as many as thirty-two or more young protozoa, each with its own capsule. It is a process of segmentation resembling what takes place in the protozoon of malaria.

We see, therefore, that in cancer we have a parasite which grows in the epithelial cell and multiplies there, and which throughout its life-history can be sharply distinguished from the cellular elements around.

These are the only elements which I regard as parasitic, and I maintain that they are essentially different from those described by Darier and Wickham, which, as I showed in another paper, I believe to be simply invaginated epithelial cells, leucocytes, &c.

My friend Professor Boyce has stated that the bodies which I describe as parasites may be nothing more than cells formed endogenously, and has, in fact, practically repeated the criticisms of Vitalis Müller, exhibiting some photographs illustrating his remarks. Professor Boyce had the kindness to show me some of his specimens, and I quite agree with him that in his sections it was impossible, for me at least, to say whether the "bodies" exhibited were parasites or not. I maintain, however, that this vagueness was due to the method of staining (hæmatoxylin), and especially of hardening (alcohol) the tissues. If Professor Boyce doubts the truth of my contention, let him fix pieces of cancer respectively with alcohol, sublimate, osmic acid, &c., and he will at once see what an astonishing difference the mode of fixation produces. Let him also examine for bacteria tissues hardened comparatively in alcohol and Foà's solution, and he will find the opposite state of affairs, the first reagent being by far preferable.

Nevertheless the criticisms of Müller, repeated by Boyce, are fair, and Plimmer and I have endeavoured to do them justice by restudying this question of the endogenous formation of cells. We quite agree that most of the descriptions given by Arnold are correct, but we do maintain that the appearances he speaks of may be explained more easily and more accurately in many other ways; and without altogether denying the possibility of the endogenous formation of cells, we say that, as far as cancer is concerned, the existence of such an endogenous formation is far from being proved, and that personally we have never seen it. In healthy tissues its existence is

more and more doubted, and the endogenous formation of red blood-corpuscles even has lately been shown to be erroneous.

Professor Boyce has unintentionally given a somewhat inaccurate interpretation of one of Arnold's figures, which he will allow me to rectify. Mr. Boyce states that "the nucleus of the included cell was often connected by fine chromatin strands with the mother-nucleus," and quotes Arnold in support of his contention. Now, if Professor Boyce will look at Arnold's plate ('Virch. Arch.,' 1883, pl. i, fig. 10), he will find that the chromatin of the so-called endogenous cells is nowhere connected by chromatin strands with the so-called mother-nucleus, and that the so-called daughter nuclei represented in Arnold's plate are, as a matter of fact, typical nuclei of leucocytes. I would also remark that Stroebe, who has repeated Arnold's work, and who has committed himself to no opinion concerning the parasites of cancer, states that he has never been able to see the formation of a daughter-cell; and that Hickson, who has put together all that has been written on the fragmentation of the nucleus in Invertebrates and Vertebrates, comes to the conclusion that "fragmentation of the nucleus only occurs where there is no cell division," a conclusion diametrically opposed to Boyce's contention.

There is one fact which, in my opinion, absolutely negatives Professor Boyce's assumption, and that is that whether one assumes that cancer cells multiply by karyokinesis, or through the fragmentation of the nucleus and subsequent endogenous formation, the life of the new cell is indissolubly connected with the chromatin of the nucleus. The first explanation which Professor Boyce should have given, therefore, is why the nuclei of the structures which he regards as endogenous cells, and which I regard as parasites, do not show the reactions of the chromatin of cancer cells, and why, when these structures divide and subdivide, they never show any of its typical reactions. And I would ask Professor Boyce to support his conclusions by giving a single example of any class of cells in the human body the nucleus of which contains no typical chromatin before, during, and after division.

Throughout this and other papers I have endeavoured to show that the bodies I regard as parasites are different from human anatomical elements in every respect; and hence I must differ entirely from the conclusions of L. Pfeiffer with regard to the structures which he considers as parasites, and which I for my

part regard as young epithelial cells and leucocytes. L. Pfeiffer himself states that his parasites are not to be distinguished from the latter structures, and from my own observations I must conclude that several things have been classed by this author under the same head. Moreover, in some cases of cancer, where we have seen a parasite lying free in the cancer alveolus, it in no way resembled a cancer cell, or leucocyte, but retained its own characteristics. To discuss the matter longer would carry me too far, especially as I have criticised these observations in another place already.

The observations which Plimmer and I have made on sarcoma are not sufficient to warrant us in publishing them, especially as we have been unfortunate in not obtaining material. We have found the subject a far more difficult one than that of carcinoma for a variety of reasons, the chief of which is the difficulty of distinguishing between a number of processes which take place in the sarcomatous tissue at one and the same time.

With regard to the ætiology of cancer through the agency of protozoa, I have nowhere made any statement on this point. In fact, I have said in several places that the cancer cells which actually contain protozoa are not, as a rule, in a state of active division. At the same time I have found nothing that distinctly negatives the possibility of these parasites standing in a causal relation to the disease, and there are some facts which are distinctly in favour of such an hypothesis.

Lastly, I should like to have the opportunity of thanking here all those who have helped me in this inquiry, more particularly Metschnikoff and Sims Woodhead; and although I have spoken in the first person throughout the paper, I may state that these opinions are endorsed by my collaborator and friend H. G. Plimmer, who quite independently of myself had recognised the presence of parasites in cancer. *March*, 1893.

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4. *On cell enclosures in cancer.*

By Rubert Boyce, M.B., and Arthur Giles, M.D., communicated by Dr. Boyce.

[With Plates VI and VII.]

In this short paper it is not our intention to review the work which has been done upon the subject of the numerous bodies found in carcinoma, and the explanation of which has proved difficult. We

have, however, in going over the literature, convinced ourselves that a large number of observers have described and figured the same curious bodies, and that it was by no means left to Soudakewitch to discover a specific body. We were fortunate to obtain from Mr. White, of University College Hospital, a carcinoma of the pancreas which presented cell anomalies and cell inclusions in unusual numbers, and we have succeeded in obtaining a large series of microphotographs. In these photographs the bodies described by Soudakewitch, Ruffer, Burchardt, and numerous others may be readily recognised, but they also bring out stages which scarcely fit in with the life history of a parasite; they, moreover, show that the cells which contain these so-called parasites are enormously hypertrophied, both as regards their chromatin and cell plasma, and that consequently we should be as much justified in discussing the "cell inclusions" as additional signs of the activity of these cells, as we should be in regarding the hypertrophy and hyperchromatosis as due to the invasion and subsequent growth of a genuine parasite.

If we go back to the early observations of Malassez we find that strong comparisons are drawn between the tumours in which these bodies are observed in man, and those met with in the familiar coccidium disease of the rabbit. We have looked in vain for strong evidence of this. The coccidium in the liver may be associated, on the one hand, with caseous and necrotic changes, and with a papillomatous and cystic condition of the bile-ducts on the other hand. But how unlike carcinoma are these simple papillomatous changes has been recently emphasised by Felsenthal and Stamm in 'Virchow's Archiv' for April, 1893. These observers agree that the coccidia may be the indirect causes of the papillomatous growths; that this is very probable the numerous observations upon other animal parasites, such as the distomata, clearly show. The finding of the cancer bodies in the metastases in man show how very different must be the mode of action of the "parasite" in man. Symbiosis must, therefore, be had recourse to, but when we pass in review the examples of this in the animal kingdom we find the parasite producing little tissue reaction; we have nothing equivalent to the phenomena observed in cancer and sarcoma. There is a great gap between the hyperplasia due to the irritative action of a parasite, and neoplasis due to symbiosis. Few would be found to deny the former, but surely

we must look for strong evidence of the latter before we accept such an interesting solution. The "parasite" of cancer has not been cultivated, and the results of inoculation are negative; reliance is therefore placed upon certain staining reactions, upon the undoubted "Eigenthümlichkeit" of the cell enclosures, and upon appearances in these bodies which have been been held to represent phases in the life history of a genuine parasite. We venture to think that it is rash, with such evidence, to so strongly maintain the parasitic nature of these bodies, and to dogmatise upon their intimate relationship to the neoplasm still more reprehensible. At the present stage we should only be concerned with the facts about these bodies, and that is the one reason we have in publishing the photographic record of our simple case. The photographs (Plates VI and VII) show a very curious series of phenomena, which evidence in the future may clearly show to belong to a parasite; in the present paper, however, we wish to demonstrate what can be said in favour of their cell origin.

The processes of cell invagination and of nuclear and cell-plasma degeneration have been discussed by those holding the parasitic view, but endogenous cell-formation must be sharply differentiated from parasitic enclosures, for the endogenous cell and the parasite are alike nucleated protoplasmic masses, and the unicellular parasites of certain mollusca, as well as those found occasionally in the alimentary tract and lungs of man, show how very difficult it is to distinguish the parasites from certain cells of the host. Failure to do so has led, on the one hand, to the mistaking of the parasites of the blood-corpuscles of the frog for active phenomena of the protoplasm of the corpuscles themselves, and, on the other hand, certain nuclear forms have been regarded as parasites (Bütschli).

Arnold has described and figured endogenous cell-formation in the giant-cells of marrow, of tuberculous glands, and of neoplasms. This phenomenon resembled closely the appearances of parasitism described by recent observers. Müller, working in Arnold's laboratory, has come to the conclusion that the bodies described by Drs. Ruffer and Walker as parasites were typical examples of endogenous cell-formation. Müller's figures and those exhibited by us agree, and their characteristic features may be thus summarised:

1. The cell enclosures are met with in abnormal cells, they are

DESCRIPTION OF PLATES VI AND VII.

(Reproductions in collotype of photographs taken by Dr. R. Boyce.)

The figures illustrate what the authors regard as varieties of endogenous cell-formation. They are taken from a case of Carcinoma of the Pancreas, and are equally magnified (Zeiss, oc. 1 mm.). (Page 216.)

PLATE VI.

FIG. 1.—Hypertrophied cell. The nucleolus is very large. There are a few scattered chromatin granules in the cell substance close to the nucleus.

FIG. 2.—In this hypertrophied cell the chromatin particle is encapsuled.

FIG. 3.—Hypertrophied cell with two nuclei and large nucleoli. The encapsuled chromatin body is much larger than in the last preparation, and presents the appearance of an enclosed cell. There is evidence of œdema of the cell-substance of the large cell.

FIG. 4.—Hypertrophied cell showing numerous encapsuled bodies of various sizes.

FIG. 5.—In this hypertrophied cell the enclosed body has the characters of a daughter cell.

FIG. 6.—The endogenous daughter cells have grown, and produced atrophy of the mother cell. Observe that one of the endogenous cells is œdematous and vacuolated, and that the other possesses a nucleus and nucleolus.

Fig. 1.

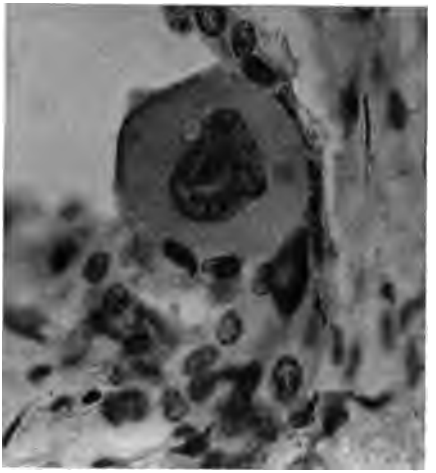

Fig. 2.

Fig. 3.

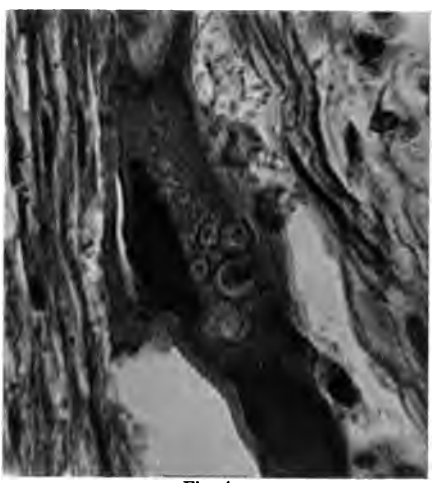

Fig. 4.

Fig. 5.

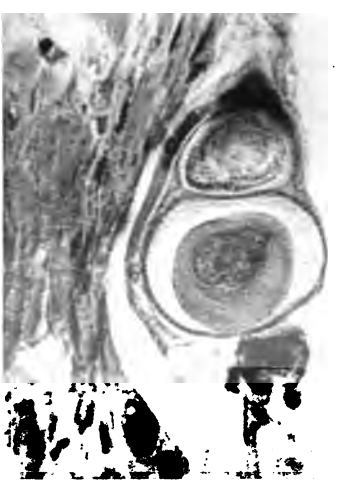

Fig. 6.

DESCRIPTION OF PLATES VI AND VII.

(Reproductions in collotype of photographs taken by Dr. R. Boyce.)

The figures illustrate what the authors regard as varieties of endogenous cell-formation. They are taken from a case of Carcinoma of the Pancreas, and are equally magnified (Zeiss, oc. 1 mm.). (Page 216.)

PLATE VII.

FIG. 7.—Hypertrophied cell. The nucleus is very large. There are two endogenous cells; one is almost liberated, the other lies in a cystic space, bounded by a deeply staining capsule. This enclosed daughter cell is nucleated and encloses a daughter cell of its own.

FIG. 8.—Mulberry form of hypertrophied cell, produced probably by the dilatation of the cystic spaces, enclosing chromatin fragments.

FIG. 9.—Cell with three enclosures.

FIG. 10.—Hypertrophied cell with marked vacuolation; some of the vacuoles have contents.

FIG. 11.—Cystic dilatation of cancer cells; the contents are granular, and have a spore-like appearance.

FIG. 12.—In this case the cystic dilatation is much greater; the enclosed cells appear degenerate.

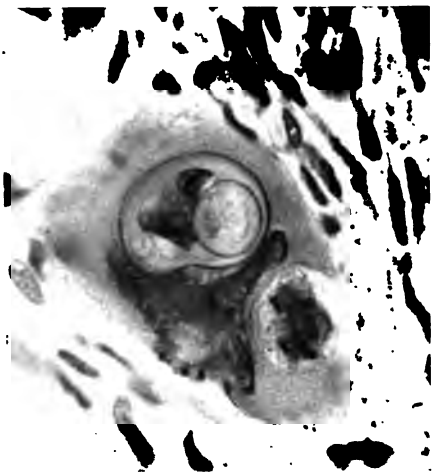

Fig 7.

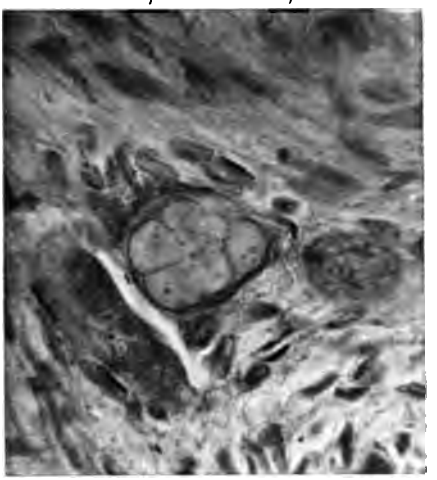

Fig. 8.

Fig. 9.

Fig. 10.

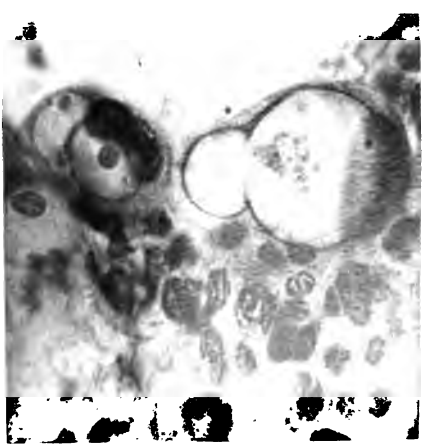

Fig. 11.

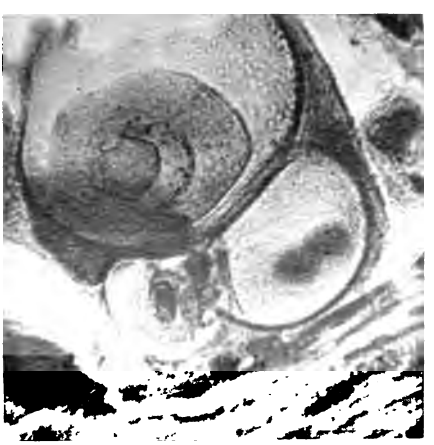

Fig. 12.

usually giant-cells; there is very often more than one nucleus; there is marked hyperchromatosis, which is characteristic of many forms of sarcoma and carcinoma cells; there are evident signs in many of the giant-cells of dropsy of the plasma. This dropsical condition is represented by a loose reticulum of the cell substance, or by the formation of groups of vacuoles.

2. The cell enclosures are single or multiple, and they usually commence in or near the nucleus, and they may occupy the place of the nucleus.

3. The enclosures consist either of little else than chromatin granules, or of a nucleus and protoplasm. The chromatin of the enclosed bodies may be broken up into several pieces, or appear as if about to divide (Steinhaus, 'Virchow's Archiv,' 1891). The chromatin may be separated by an enclosing vacuole from the rest of the cell, and often stains like the nucleolus of the mother-nucleus.

4. There is a capsule; this is regarded by Dr. Ruffer as formed by the cancer cell itself, and with this we agree. Flemming showed that the capsule of the fat-cell may be highly differentiated and stain characteristically.

5. The capsule expands either by the growth of the enclosed cells, or by the accumulation of fluid. The capsules may, in fact, contain perfect cell enclosures, large or small granular masses, round homogeneous bodies, fine granular *débris*, or only fluid. Compare the changes in the nuclear substance in molluscum contagiosum.

6. When several capsules are formed in the same cell, or in close proximity, a mulberry mass results, comparable to the mulberry fat-cells (derived from one cell) of certain of the lower animals. The partitions may rupture and fusion may occur.

7. Capsules are met with enclosing more than one nucleated body, or two or more perfectly formed cells; the enclosures themselves may be cystic, and enclose other bodies.

8. There is not the least trace of irritation or leucocytic reaction in the neighbourhood of the cell enclosures.

Finally, cancer bodies have been freely compared with, and called sporozoa, but cancer bodies and sporozoa are in marked contrast in the following points:

1. The intra-cellular sporozoa (sarcosporidia) seem to be of a harmless nature, not causing inconvenience to their host (Bütschli), but rather living on terms of symbiosis.

2. Sarcosporidia are nearly always large, and often visible to the naked eye.

3. The cystic or resting stage of sporozoa is the only one represented at all definitely among cancer bodies; the active amœboid stage, pseudopodial or ciliated forms, as observed in coccidia and klossia, are never seen.

4. The numerous round, lenticular, or falciform spores are not seen in cancer in man.

5. The bodies which are compared to spores are clear, like fat-globules, and of unequal size; the spores of sarcosporidia are invariably of uniform size, and generally granular.

6. The spores of gregarinidæ always have a siliceous, or chitinous, and well-marked capsule. *March 7th*, 1893.

B. THE ANTI-CHOLERA VACCINATION: AN EXPERIMENTAL CRITIQUE.

By E. Klein, M.D., F.R.S.

Having for some time been engaged for the medical department of the Local Government Board in investigating the antagonistic action of various pathogenic bacteria, I have during the last year extended these investigations to the anti-cholera vaccination, as described by M. Haffkine, of the Pasteur Institute. Haffkine, in an extensive series of experiments, has shown that if a large dose of choleraic comma bacilli, grown on, and then removed from, the surface of solidified nutrient agar, be injected (as a suspension in sterile beef bouillon) into the peritoneal cavity of a guinea-pig, a fatal result is produced in, or within, twenty-four hours, the peritoneal cavity containing fluid exudation full of the living comma bacilli. R. Pfeiffer[1] had already shown that this result is produced by injecting intra-peritoneally large masses of the cholera bacilli, either living or after having been previously killed by heat or drying, by chloroform, ammonium sulphate, alcohol, or other means; and he justly concludes from these experiments that the comma bacilli themselves contain in their protoplasm a poisonous

[1] 'Archiv f. Hygiene und Infectionskrankheiten,' vol. xi, pt. 3, p. 393.

principle. The fatal result in the guinea-pig thus obtained with large doses of the cholera bacilli was shown to be due to acute severe peritonitis, and it was further shown that the same result is obtained by the same methods with the vibrio of Finkler and the vibrio of Metschnikoff, so that these two species also include in their substance a similar poisonous principle. Notwithstanding these facts, Pfeiffer regards the intra-cellular poison of the cholera bacilli as "the primary cholera poison."

Haffkine starts from these experiments, and finds that by transmitting, by intra-peritoneal injection, the (peritoneal) exudation of a guinea-pig dead after the above injection through a succession of guinea-pigs (twenty to thirty generations), an exudation fluid is ultimately obtained, of which the cultivation on agar possesses a high degree of virulence—"virus fort;" so much so that a small dose of this cultivation injected intra-peritoneally into the guinea-pig produces a fatal result in eight to twelve hours, the fatal dose of this "virus fort" being in amount only a fifth, a sixth, or less of that required of the original cholera culture. Repeated subcutaneous injections of small doses of this "virus fort" produce in the guinea-pig a transitory constitutional illness; at the seat of inoculation there appears a soft œdematous swelling, which, after a few days, diminishes in size, becoming at the same time firmer, and is either totally absorbed or more generally produces a local necrosis and ulceration of the skin, which ultimately completely heals up. Guinea-pigs which have been twice thus subcutaneously inoculated with small doses of the "virus fort," generally attenuated by the addition of phenol, prove themselves refractory—that is, "vaccinated"—against the intra-peritoneal injection of a fatal dose of the "virus fort," that invariably kills control guinea-pigs in eight to twelve hours. I have made a considerable number of experiments on guinea-pigs in a similar direction; the results of these I now propose, with the sanction of the Medical Department of the Local Government Board, to communicate to the Society. These results may be arranged in the following order:

1. Experiments of intra-peritoneal injection were made with beef bouillon suspension of various microbes, grown on the surface of solidified nutrient agar; the amount used for each animal being in all cases one fifth to one half of a culture tube, each culture tube presenting an agar surface of 6 cm. by 2 cm. The microbes used were—(*a*) the cholera bacillus, (*b*) the vibrio of Finkler, (*c*) the

bacillus coli, (*d*) the proteus vulgaris, (*e*) the bacillus prodigiosus, and (*f*) the bacillus of typhoid fever.

2. In all cases the intra-peritoneal injection of one fifth to one half of a culture was followed after a few hours (four to eight) by distinct illness; the temperature is lowered, the animals are quiet, do not move and do not feed; after about eight to twelve hours, sometimes earlier, this condition is more pronounced; next day, that is within twenty to twenty-four hours, the animals as a rule are found dead. On *post-mortem* examination there is found intense peritonitis with pseudo-membranes on the omentum and liver; the intestine and particularly the serous covering of the stomach are much congested. The small intestine is either relaxed and filled with sanguineous mucus, or is more or less contracted. The peritoneum contains more or less turbid, slightly sanguineous fluid exudation filled with the microbes used for the injection. The heart's blood also contains the microbes, as cultivations prove; the colonies obtained by such cultivations are always fairly numerous, but of course not so numerous as those of the peritoneal exudation. It must be insisted on, that in all this there is absolutely nothing to warrant the assertion that the results produced by the cholera bacillus are comparable to cholera. The symptoms of the disease and the pathological changes produced in the guinea-pigs by the intra-peritoneal injection are for all the above species exactly the same, but it ought to be mentioned that the cultivations of bacillus coli, bacillus of typhoid, and bacillus prodigiosus act more virulently than those of the cholera bacillus or the vibrio of Finkler, since the dose of the latter, in order to be fatal, has to be larger than that of the former, and the appearances produced by the same amount of culture, though identical in character, are more intense in the case of the bacillus coli and typhoid, and bacillus prodigiosus, than in that of the vibrio of cholera and of Finkler respectively.

3. The same result is also produced by the intra-peritoneal injection of sterilised culture (exposure to 62° to 65° C. for ten to twelve minutes), the amount required to produce a fatal result being, however, a little larger than that required with the living bacilli.

4. As only the bacteria themselves (scraped off from the surface of the agar culture) are used for these injections, it follows that all these six different species of bacteria contain in their protoplasm a poisonous principle, which appears to be the same for all species,

since the disease produced and the anatomical changes found on *post-mortem* examination are in all cases identical.[1]

5. Non-fatal doses (one fifth to one tenth of a culture) injected intra-peritoneally produce a transitory illness; after recovery the guinea-pigs are found refractory against the action of otherwise fatal doses of the same species injected intra-peritoneally—that is to say, doses which are fatal to control guinea-pigs. This refractory condition is obtained by non-fatal doses both of the living and of the sterilised cultures.

6. The refractory condition produced by intra-peritoneal injection of a non-fatal dose of one of the above six species holds good against all the other five; thus guinea-pigs after surviving the intra-peritoneal injection of non-fatal doses of vibrio of Finkler, of proteus, of bacillus coli, of bacillus prodigiosus, or of typhoid bacillus respectively, are found refractory against a fatal peritoneal dose of cholera bacilli. I have experimented with a considerable number of guinea-pigs which had survived the peritoneal injection of non-fatal doses of the vibrio of Finkler, of the bacillus coli, and bacillus prodigiosus respectively; on a second intra-peritoneal injection with fatal doses of the cholera bacilli—nay, even of Haffkine's "virus fort," they proved themselves completely protected. These experiments furnish a further confirmation of the above conclusions —namely, that the intra-cellular poison of these different bacterial species is identical, since they mutually protect the animals.

7. The subcutaneous injection of cultures of all these different species into guinea-pigs produces the identical disease; a transitory general illness, a local soft œdematous swelling, which after a few days diminishes in size and becomes firm, and either altogether becomes absorbed or leads to local necrosis and ulceration of the skin; the latter only takes place after injection of large doses of the culture. The effect produced by bacillus coli, bacillus prodigiosus, and bacillus of typhoid is a little more intense than that produced by the others; living culture is more virulent than sterilised culture, but in its general characters it is the same for both.

8. Just as is the case in Haffkine's experiments with the cholera

[1] The similarity of the results obtained by Roux and Rodet after intra-peritoneal injection into guinea-pigs of cultures of bacillus coli and bacillus of typhoid fever do not therefore prove anything as to the relationship of these two species, since the identical results are obtained with the proteus, bacillus prodigiosus, cholera vibrio, and vibrio of Finkler.

vibrio, so also with the vibrio of Finkler, the bacillus coli, the bacillus of typhoid, and the bacillus prodigiosus, the intra-peritoneal transmission through a series of guinea-pigs produces a marked increase in the virulence of the peritoneal exudation. In the case of the bacillus coli, bacillus prodigiosus, and bacillus of typhoid, already after two transmissions the subcutaneous injection of a few drops of the peritoneal exudation, or of a culture grown from this, produces a fatal effect; whereas a considerably larger dose of the original culture fails to do more than produce a local tumour. In the case of the vibrio of Finkler, also, the transmission of the peritoneal exudation through a few successive guinea-pigs produces an exudation, of which a few drops subcutaneously injected produce a pronounced local tumour, leading to necrosis and ulceration of the skin, such as is only produced by large doses of the original culture.

9. Animals which have been repeatedly injected subcutaneously, either with living or with sterilised cultures (all from the surface of solid agar), prove refractory against the intra-peritoneal injection of fatal doses of the living cholera cultures. I have a number of guinea-pigs which have been injected subcutaneously on several occasions with sterilised cultures of vibrio of Finkler, of bacillus coli, and of bacillus prodigiosus. They were, after the lapse of ten to twelve days, subjected to intra-peritoneal injection with fatal doses of Haffkine's "virus fort," but they proved themselves refractory. I may mention one such series of experiments: (*a*) Six guinea-pigs were subcutaneously injected with sterilised (62° C. for ten minutes) cultures of vibrio of Finkler on four occasions (one fifth of a culture tube being used for each animal on the first, third, fourth, and sixth days). Twelve days after the last injection they were injected intra-peritoneally with Haffkine culture of "virus fort," one twelfth of a culture of this latter being used for each animal. Two control guinea-pigs injected at the same time with one twelfth culture of this same "virus fort" were found dead the next morning, but only one of the above six Finkler guinea-pigs died, after about thirty hours; the other five remained alive. (*b*) Four guinea-pigs were subcutaneously injected with sterilised culture of bacillus prodigiosus on four occasions (one fifth of a culture tube being used for each on the first, third, fourth, and sixth days). Twelve days after the last injection they were each injected peritoneally with one twelfth of a Haffkine culture of "virus

fort." All remained alive. (*c*) Four guinea-pigs were subcutaneously injected on three occasions (one-fifth of a culture tube being used for each animal on the first, second, and fourth day) with sterilised culture of bacillus coli. Twelve days after the last injection they each received into the peritoneum one twelfth of a culture of the same Haffkine's "virus fort." All four guinea-pigs remained alive.

10. The different species of microbes thus experimented with, including the cholera vibrio, contain in their substance the same poisonous principle, but this must be carefully distinguished from the poisonous substances (ptomaines, specific toxins, and others) which are elaborated by them in nutritive media, including the animal body. While the intra-cellular poison, as is shown by the above experiments, appears to be identical for all the above species (judging from their identical action on the guinea-pig after subcutaneous and intra-peritoneal injection and from their mutual protection), the latter, that is the specific toxins produced in the nutritive media, differ (*a*) from one another and (*b*) from the intra-cellular poisons. As regards (*a*) this is well known; as regards (*b*) several cases in point may be mentioned. Tuberculinum of Koch contains the intra-cellular poison of the tubercle bacilli; as is well known, it produces on subcutaneous injection into tuberculous individuals a definite phlogo- and pyo-genic reaction, but when the tubercle bacilli grow in the body of a suitable animal, they produce additional pathological processes, namely, the characteristic giant-cell formation, the spreading necrosis and caseation. Or take the proteus vulgaris: the effect of large doses of the bacilli themselves after intra-peritoneal and subcutaneous injection, particularly the latter, have been described above; these effects are different from those resulting from the subcutaneous injection of small doses of the ptomaines produced by the proteus growing in albuminous matter.

Again, in the case of the diphtheria and tetanus bacilli, the well-known effects produced on inoculation of these bacilli are those of the specific toxins elaborated by them at the seat of inoculation and absorbed by the system, not of the bacilli themselves—*i. e.* not of the intra-cellular poison—since these microbes do not live in the blood or tissues of the infected bodies.

A further consideration to be added here is this: When the action of an agar culture of the above-named six species is compared with that of the peritoneal exudation even of the first

guinea-pig in the series, it will be found that the latter fluid is much more virulent, and considerably smaller doses produce a fatal result than in the case of the former. The explanation, I presume, is this: In the case of the agar culture the action is chiefly that of the intra-cellular poison, while in the case of the peritoneal exudation there is in addition the action of the ptomaines or toxins that had been elaborated by the bacilli in the peritoneal fluid. For the same reason also the fatal dose of the living agar culture injected into the peritoneal cavity is smaller than that of the sterilised culture, as has been stated above, for in the latter the effect produced is only that of the intra-cellular poison injected, whereas in the former must be added also the action of the poisons elaborated in the peritoneal fluid by the living bacilli.

That there exists a notable difference between the intra-cellular poison and the toxins elaborated by the bacilli in the culture medium is strikingly shown by the fact that protected guinea-pigs which have survived the intra-peritoneal injection of fatal doses of Haffkine's "virus fort," mentioned on former pages, when subsequently inoculated peritoneally with gelatine culture (liquefied by the growth) of the cholera bacilli promptly succumb; thus proving that, though protected against the intra-cellular poison, they are by no means protected against the toxins elaborated in the culture medium (gelatine) by the bacilli.

11. It follows from all this that if in any case a refractory condition against the intra-cellular poison is established, it does not follow that this refractory condition necessarily holds good also against the specific toxins, or against the growth and multiplication of the bacilli, and the production by them of their specific toxins in that refractory body. As we have shown, the previous injection of the intra-cellular poison of the vibrio of Finkler, of the bacillus prodigiosus, or of the bacillus coli, is capable of rendering the body refractory against the intra-cellular poison of the cholera bacillus, or of the typhoid bacillus, but it would not be justifiable to assume that such a refractory condition is an index of insusceptibility against natural infection with cholera or typhoid fever; as a matter of fact, guinea-pigs protected against the intra-cellular poison of the cholera bacilli succumb to the toxins produced by these in the culture medium.

March 21*st*, 1893.

XI. DISEASES, ETC., OF THE LOWER ANIMALS.

1. *Septic endocarditis in a calf.*

By H. Betham Robinson for Dr. Sydney F. Wright.

The calf presenting the above lesion was a twin. The other twin is alive at the present time, and has never shown any signs of illness.

The calf was born at the beginning of January, 1893, and lived five weeks.

The first symptoms of illness were noticed on the fourth day, and about a week later, shortness of breath, fainting fits, and preference for the recumbent position were observed. The attacks of syncope often came on while the animal was in the act of sucking, and placing it on its legs almost always evoked an attack. In the end it fell down dead with blood coming from the mouth and nostrils.

At the *post-mortem* the heart showed vegetations at all the orifices, the right side being especially affected. In the lungs were large hæmorrhagic areas with patches of solid lung around. The kidneys showed characteristic wedge-shaped infarcts, one or two of them breaking down. There were no abscesses in other viscera, skin, or brain.

The few joints examined presented no abnormal appearances, neither did the pleura, peritoneum, or pericardium.

The condition of the umbilicus and umbilical vein was not observed.

The case was evidently a septic one, and as the right side of the heart was chiefly affected, it may be surmised that probably the unexamined umbilicus and veins were responsible for its origin. It is curiously to be noted that part of one of the placentæ was

retained for three days, and when passed was very offensive. The cow herself was evidently very ill during this time, but quickly recovered after the expulsion of the placenta. *May 16th*, 1893.

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2. *Hyaline fibroma of a cow's udder.*

By Dudley Cooper.

During life the tumour was noticed beneath the skin over the udder of a cow which was being used for experimental purposes. The tumour was of about the size of a horse chestnut, very hard, not adherent to the skin, fairly moveable, and situated close to the base of the left posterior teat.

After death the tumour, which measured an inch and a half in its longest diameter, obviously consisted of some very dense tissue in which small gelatinous-looking areas could be seen on section.

On microscopical examination the growth was composed of a dense fibrous tissue with the fibres grouped together in bundles of various sizes, like that met with in chronic interstitial mastitis. Very few cellular elements were to be seen, and those that were present were met with mostly at the periphery. The fibrous tissue had in many places become transformed into a translucent homogeneous substance, staining deeply with borax-carmine, and probably of a hyaline nature. This change, which has been well described in fibromata occurring in the human subject by Klebs [1] and others, was more marked towards the centre of the growth. There was no indication of any granular structure and very few blood-vessels were to be seen.

No reference to such tumour occurring in connection with mammary glands is to be found in any of the best continental or English text-books on veterinary science, although fibromata in other parts of the body are stated to be not infrequently met with in animals. *December 6th*, 1892.

[1] Klebs, 'Allgemeine Pathologie,' ii.

3. *Adeno-fibroma of mamma in a male pied rat.* (*Card specimen.*)

By Samuel G. Shattock.

The tumour, which is of flattened spheroidal form and one and a half inches in diameter, was removed without difficulty from beneath the skin of the right side of the thorax in an old pied rat.

In section it presents to the naked eye much the appearance of pancreas, and is made up of small tough lobules, held together by connective tissue. Here and there occur small cysts filled with intra-cystic growths.

Histologically the tumour has all the characters of a typical adeno-fibroma of the human mamma, and consists of dense fibrous tissue disposed in lobules, which are conjoined into a single mass by a looser connective tissue. In the lobules lie groups of acini, arranged precisely as in the human subject, and lined with spheroidal epithelium.

There are, in the fibrous tissue, considerable numbers of large oval or polyhedral, tailed, or branching, granular connective tissue corpuscles, the fine granules in the cell-bodies of which are intensely stained with the hæmatoxylin. *May 2nd*, 1893.

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4. *Parasitic cysts from a rabbit.*

By H. Betham Robinson, M.S.

The rabbit from which the cysts were taken died during a physiological experiment. At the *post-mortem* it was found to be very diseased.

Among the muscles of the back legs were several cysts, two especially being very large. In the subcutaneous tissue of the back others were met with. There were one or two whitish nodules in the liver, and the omentum and mesentery were studded

with white nodules. The whole condition was supposed to be psorospermial, but investigation proved them otherwise.

The cysts on section showed a fairly marked adventitia from the connective-tissue around; inside this was a whitish soft membrane with small pear-shaped bodies projecting into the interior. There was a clear watery fluid in the interior which was albuminous. The pear-shaped bodies were inverted scolices, their heads being inside the pear-shaped bodies. The heads on examination had two rows of hooklets, one of larger and one of smaller size. In some specimens examined, four suckers were found, and in others, six; the latter condition was probably some abnormality.

The cysts on the peritoneum proved to be of the same nature.

The parasite seemed to correspond to the cystic form of Tænia serrata of the dog; that is, assuming that the multiplication of suckers was an abnormality. In the works on the subject by Kuckenmeister, Cobbold, and others, no mention is made of such a condition. *November* 1*st*, 1892.

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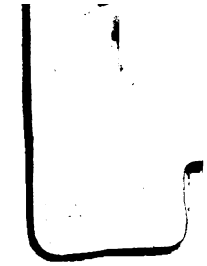
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